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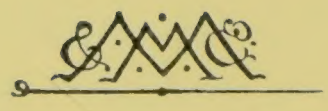
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SYSTEM OF MEDICINE

BY MANY WRITERS

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VOLUME III

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PREFACE

THIS volume has undergone very considerable rearrangement, and in the main, especially as regards the more important articles, is a rewritten rather than a revised successor of Volume III. of the original edition. Dr. Garrod's articles on Rheumatoid Arthritis, Osteo-Arthritis, and other forms of joint affections are entirely new, not only in arrangement but in substance; while the clear distinction drawn between Rheumatoid Arthritis and Osteo-Arthritis breaks fresh ground, at any rate as regards systematic treatises in this country. A new article on Pulmonary Osteo-Arthropathy has been provided by Dr. Batty Shaw; and Dr. Poynton, who now collaborates with Dr. Cheadle in the article on Rickets, has contributed a separate account of Achondroplasia. The late Sir William Roberts's article on Gout and the late Dr. Ralfe's account of Diabetes Insipidus have been revised by Prof. Rose Bradford.

Among the diseases of the alimentary canal the article on Diseases of the Mouth has been entirely rewritten by Mr. Walter Spencer; a new article on Congenital Hypertrophy of the Pylorus has been contributed by Prof. Still; and the three important articles on Appendicitis, Intestinal Obstruction, and Visceroptosis, formerly contributed by Sir F. Treves, have been rewritten by Mr. Lockwood, Mr. Barnard, and Dr. A. Keith respectively. A fresh section on the difficult subject of the Bacteriology of Diarrhoea has been written by Dr. Slater; and the Differential Diagnosis of Diseases of the Anus and Rectum, originally contributed by the late Mr. Herbert Allingham, has been revised by Mr. Mummery, who has introduced a coloured plate shewing various morbid appearances

as displayed by the sigmoidoscope. Among the articles to which considerable additions have been made, that on Gastric Ulcer may be specially mentioned as one which in any circumstances would have commanded attention, but now may be the last memorial of its distinguished author. This is not the place to do full honour to the memory of Prof. Dreschfeld; here it can only be said that in him we lament the death of a colleague whose scientific methods, clinical experience, and large and accurate learning placed him in the first rank of contemporary physicians.

The section on Diseases of the Peritoneum is prefaced by an account of Shock by Dr. T. G. Brodie. The important subject of Acute Peritonitis, to which Sir F. Treves made such a valuable contribution in the former edition of this *System*, is now dealt with by Dr. Allchin, and by Dr. F. W. Andrewes, who has given a clear description of the general pathology and bacteriology of the disease. Lastly, an entirely new and more comprehensive article on Subphrenic and other forms of Peritoneal Abscess by Dr. Acland takes the place of the original account of Subphrenic Abscess by the late Dr. Lee Dickinson.

T. C. A.
H. D. R.

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In order to avoid frequent interruption of the text, the numbers indicative of items in the lists of "References" are only inserted in cases of emphasis, where two or more references to one author are in the list, where an author is quoted from a work published under another name, or where an authoritative statement is made without mention of the author's name. In ordinary cases an author's name is sufficient indication of the corresponding item in the list.

GENERAL DISEASES OF OBSCURE CAUSATION

RHEUMATOID ARTHRITIS

OSTEO-ARTHRITIS

SPONDYLITIS DEFORMANS

ARTICULAR LESIONS OF INFECTIVE DISEASES

Gonorrhoeal Rheumatism; Dysenteric, Syphilitic, Pneumococcal Arthritis; Rhumatisme Tuberculeux

INTERMITTENT HYDRARTHROSIS

PULMONARY OSTEO-ARTHROPATHY

OSTEITIS DEFORMANS. Leontiasis Ossea

MOLLITIES OSSIUM. Multiple Myeloma

RICKETS

ACHONDROPLASIA

GOUT

DIABETES MELLITUS

DIABETES INSIPIDUS

SEA-SICKNESS

MOUNTAIN-SICKNESS

RHEUMATOID ARTHRITIS, OSTEO-ARTHRITIS, ARTHRITIS DEFORMANS

By A. E. Garrod, M.D., F.R.C.P.

It is no longer possible to look upon the cases usually included under the names of rheumatoid arthritis, osteo-arthritis, and arthritis deformans, to mention only a few of the designations applied to them, as examples of a single disease, which, although it appears under somewhat varied clinical aspects, is accompanied by a group of anatomical changes in joints which conform to the classical description of the osteo-arthritic lesions. It is becoming more and more evident that the clinical and anatomical groups do not coincide; that among the cases included in the clinical group there are many in which the osteo-arthritic changes are not present, and that by no means all the cases in which such changes occur conform to a definite clinical type.

The conditions under consideration are so seldom fatal that the morbid anatomist has very few opportunities of investigating the condition of the joints in the more active stages, and as a rule can only observe the havoc wrought in structures which have, in the past, been seats of long-standing disease. It is probable that the lesions seen are determined as much by the nature of the structures attacked, as by the processes in which they had their origin. It is possible, indeed, that what are known as the osteo-arthritic lesions are in no way specific, but may occur in joints which have been the seats of various forms of disease, and may even be provoked by traumatic influences acting during the degenerative period of life. Hence the work of classification of the several conditions included under the names quoted must be carried out mainly on clinical lines, but we may hope and even expect that in the future bacteriology will contribute largely to the attainment of an accurate grouping of them.

The realisation that we are here dealing with a group of maladies and not with a single disease is in itself a great advance, but the progress made in the direction of reconstruction is as yet scanty. This is due in no small measure to the great difficulty of so describing the clinical features of articular lesions as to convey to others a clear mental picture

of the special class of cases which it is intended to describe, although to the describer the distinctive features are sufficiently obvious. No one who has studied the more recent literature of the subject can fail to realise that this is a very real obstacle to progress. Some of the classifications proposed are too elaborate to carry conviction, and some of them presuppose a knowledge of the pathology of the conditions classified to which we cannot as yet lay claim. The great bulk of the cases spoken of as examples of rheumatoid or osteo-arthritis would seem to be examples of two maladies which present distinct clinical features, and in the present article these maladies will be discussed under separate headings. Since what is least of all to be desired is further complication of nomenclature where names are already far too many, it will be convenient to speak of the two classes of cases under the old names of rheumatoid arthritis and osteo-arthritis respectively, but it may be mentioned that they conform closely to the atrophic and hypertrophic arthritides of Goldthwaite, whose classification is adopted by other American authors. Moreover, the disease here spoken of as osteo-arthritis is the arthritis deformans of German writers. Among English writers upon the subject Dr. Bannatyne, although he does not completely separate the two groups of cases here described, suggests that they really represent distinct disorders.

It cannot be asserted that the rheumatoid arthritis of this classification is altogether a pure category. In it are, doubtless, still included cases of infective arthritides of various kinds, and it is an open question to what extent the cases met with in children are to be regarded as examples of the same malady as is seen in adults. That some are of this nature cannot, I think, be doubted, but this cannot be asserted of all or perhaps of the majority. On the other hand, the majority of the adult cases are so uniform in feature that it can hardly be doubted that they constitute a definite morbid species, and it is unlikely that a description of their clinical aspects will be seriously blurred by the intrusion into the material upon which it is based of cases of extraneous kinds. Among such extraneous cases are almost certainly included some of rheumatic origin, and more or less chronic arthritic lesions due to the invasion of the joints by known specific bacteria, such as the gonococcus, and possibly the pneumococcus, although the arthritis produced by this last organism is usually acute and suppurative. To disentangle such cases from the rest is no easy task, and is sometimes wellnigh impossible when the active disease is over. In some instances a clear history of a primary infection, such as a gonorrhoea, is forthcoming, in others the clinical course of the malady arouses suspicion by unusual features, and in others again the local treatment of an infective focus, such as an ulcer of the rectum or pyorrhoea alveolaris, suffices to arrest the malady and may even be followed by rapid recovery.

Again, among the cases of the osteo-arthritic group, sharp clinical divisions have to be made, and such varieties as the hip-joint disease of elderly people and Heberden's nodes call for separate description. With

these it will be convenient to include the remarkable malady known as spondylitis deformans, although the true place of the disease or diseases so styled is still very obscure and is probably elsewhere.

Rheumatoid Arthritis

Whether it be acute, subacute, or insidious in its onset the course of rheumatoid arthritis is apt to be lengthy and relentless. From time to time fresh joints are affected, until in the most severe cases hardly an articulation in the body escapes. The joints attacked become the seats of a fusiform enlargement, which is chiefly due to thickening of their capsules and is not attended, in the active stages at least, by any lipping of the bones or osteophytic outgrowths. When the active stage is at an end the damage wrought in the articular structures and, above all, the permanent contracture of the wasted muscles, tend to produce deformity and crippling of a most grave kind. Yet, since the viscera are seldom affected, the disease has little tendency to shorten life, although it is apt to deprive the patient of much that lends value to life.

Etiology.—The disease may begin at any age, for it is hardly possible to doubt that some at least of the cases of persistent arthritis met with among children belong to this group, and on the other hand it may arise in the later decades of life, at periods when the characteristic changes of osteo-arthritis would naturally be expected. The period of greatest liability is between the ages of twenty and forty years.

The far greater liability of females to rheumatoid arthritis is unquestionable, and in this respect statistics fully confirm the impression derived from daily experience. The published statistics do not discriminate between cases of rheumatoid and osteo-arthritis, and are accordingly not available for our present purpose, but the data as to the sex and age incidence of the malady under discussion are clearly brought out by the following figures drawn from a series of 100 cases. These cases were taken without selection save that all were excluded which did not exhibit the special features of the disease here spoken of as rheumatoid arthritis.

Age at Commencement.

	0-10.	10-15.	15-20.	20-30.	30-40.	40-50.	50-60.	60-70.	Totals.
Females . .	1	3	3	22	28	14	5	1	77
Males . .	0	0	0	7	6	3	7	0	23
	1	3	3	29	34	17	12	1	100

As it was necessary in most cases to rely upon the patients' own

statements as to the age at onset of the troubles, some unavoidable error must have crept in.

Any condition which tends to lower the standard of health may act as a disposing cause of rheumatoid arthritis — as of other diseases more certainly known to have a bacterial origin. Among such disposing causes may be mentioned periods of worry and anxiety, the nursing of sick friends, work under non-hygienic conditions, and lowering maladies of various kinds, amongst which a prominent place must be assigned to influenza. From an attack of influenza many patients date the commencement of their arthritic troubles. As has long been recognised and as was specially emphasised by the late Dr. Ord, disorders of the genital organs may play an important part in determining the far greater liability of the female sex to rheumatoid arthritis. In one well-defined class of cases the onset of the disease follows at a short interval after parturition, or after a miscarriage, and it is difficult to avoid the conclusion that in these cases the uterus has been the point of entry of an infection. In others again menorrhagia and other uterine disorders appear to act as disposing causes, just as they may aggravate the gravity of the disease when once established. Of recent years stress has been laid upon infective foci of various kinds as originators of rheumatoid arthritis, and in a small proportion of cases such foci are to be found, especially pyorrhoea alveolaris; affections of the naso-pharynx and ulcerated piles may also be mentioned. Moreover, in some cases treatment directed to such a focus has brought about rapid amelioration, or even complete recovery of the inflamed joints. It is doubtful, however, whether these cases are really examples of the specific disease rheumatoid arthritis, assuming that such a specific disease exists, and not examples of other forms of infective arthritis which mimic it more or less closely. In the great majority of instances careful inquiry and examination, which should never be omitted, fail to reveal any local focus of infection, and the onset of the rheumatoid arthritis is as little capable of explanation as that of most attacks of rheumatic fever. Even disposing causes are often not to be traced, and the malady commences at a time when the patients believe themselves to be in perfect health.

Morbid Anatomy.—Whereas our knowledge of the morbid anatomy of osteo-arthritis is very complete, the material upon which to base a description of the changes found in undoubted cases of rheumatoid arthritis in the active stage, as distinguished from the stage of sequels, is very scanty. Nor is this to be wondered at when the rarity of a fatal ending in this stage is considered.

The following description is mainly based upon the observations recently recorded by Dr. Hale White on a young woman under his care whose death was due to an intercurrent malady. There was much fibrous thickening outside the joints, with thickening of the ligaments. The synovial membranes were swollen and of a pinkish-red colour. The articular cartilages were, in the main, unaffected, but there was a very slight erosion of the cartilage covering the external condyle of the right

knee. Synovial fluid was present in excess. There were no outgrowths from the bones nor from the edges of the cartilages. In a word the disease was not one of bones and cartilages, but of the synovial membranes and periarticular tissues. Microscopic sections cut from a proximal interphalangeal joint, after decalcification of the bones, shewed some thinning and erosion of the edges of the cartilage apparently due to pressure of the swollen synovial fringes. The cancellous tissue of the bones was more open than usual, and foci of inflammation were seen, with small-celled infiltration, and a relative absence of fat-cells. These bone changes, however, were of an altogether slighter kind than those in the synovial membranes. Dr. Bannatyne describes a great development of new-formed tissue in the soft parts in acute cases, and a general softening of the cartilages, with here and there an erosion; the bones as red, rarefied, vascular, and soft; the synovial membrane as bright red from injection, and in the later stages hypertrophy of the villi which may have undergone cartilaginous or fatty changes, with the formation of dendritic fringes. Max Schüller has made a detailed study of the process of formation of the synovial villi, lipoma arborescens, from the normal synovial fringes. Although the formation of such villous outgrowths is very general in rheumatoid arthritis, and, as in Dr. Hale White's case, their presence may lead to absorption of the cartilages in places, it is only in exceptional cases that they attain to the extreme degree described by Schüller, and become responsible for a conspicuous and irregular distension of the synovial capsule, with corresponding swelling of the joint. The villous processes, which, when seen during life, have a deep red or bluish-red colour, assume various shapes, some being slender and thread-like, others tortuous and flattened. They may spring from all parts of the synovial membrane, or may be confined to certain definite regions. Schüller describes the synovial fluid as scanty in the villous cases, and never purulent. The cartilages may be intact.

In all the descriptions of the condition of the joints in the more acute stages of rheumatoid arthritis there is an absence of the osteophytic outgrowths and of the erosion of the central portions of the cartilages which are pronounced features of osteo-arthritis. Such damage to the cartilages as is seen appears to be merely a secondary result of the pressure of hypertrophied synovial fringes. Max Schüller strongly emphasises these differences, and draws a sharp distinction between the cases which he describes and examples of arthritis deformans, which term he would only apply to cases in which the characteristic osteo-arthritic lesions are present.

In cases of old standing, which come to examination when the active stage is long past, there may be complete destruction of the articular structures; firm fibrous bands surround what were once the articular cavities, the cartilages may be entirely destroyed, and when fixation has been complete bridges of cancellous bone may stretch across from one surface to another. There may be some proliferation of bone, with the

formation of sharp spicules, which are very unlike the rounded osteophytes of osteo-arthritis.

Bacteriological Investigations. — Several observers have found bacteria in the synovial fluid and synovial membranes of sufferers from rheumatoid arthritis. The earliest observations were those of Max Schüller upon cases of the villous form of the disease, of which he has made a special study. The organisms were for the most part met with in the tissues, such as the inflamed synovial membrane and the hypertrophied fringes. Both bacilli and cocci were found, but Schüller attaches specific importance to the bacilli alone. These were constantly present, whereas the cocci were only occasionally met with, and especially in joints which were the seats of the ankylosing, atrophic form of the disease. The cocci, which resembled the ordinary pyogenetic micrococci, Schüller regards as evidence of a secondary infection, such as is not uncommon in gonorrhoeal rheumatism and other forms of infective arthritis. The bacilli were short and thick, with polar staining. Their characters were in the main uniform in the different cases examined, such differences as were presented being attributable to differences of media and different stages of growth. Injection of the bacilli into the joints of rabbits was followed in the course of two or three months by a villous arthritis which reproduced in miniature the condition of the human joints from which the bacilli had been isolated.

In 1896, Drs. Bannatyne, Wohlmann, and Blaxall described a micro-organism which was almost always found in the joints of patients with true rheumatoid arthritis as here described, but which they failed to obtain from osteo-arthritic joints, or from joints which were the seats of other forms of disease. The organisms—which were very minute, about 2 μ in length,—although at first sight resembling diplococci proved to be bacilli with bipolar staining. The same bacillus was found in the blood in some cases, and in fragments of synovial membrane. The organism grew in peptone broth, and also grew upon agar in minute highly transparent colonies which formed a transparent film on the surface of the medium. Aniline methylene blue proved the most satisfactory stain. With Gram's method it was almost completely decolorised. Inoculation experiments yielded no conclusive results. Soon afterwards Chauffard and Ramond found in a single case a small Gram-staining diplo-bacillus, which they succeeded in cultivating in synovial fluid only. In this medium it grew readily. Inoculation experiments were not attended by any success. A case recorded by von Dungern and Schneider, which is sometimes quoted in this connexion, was apparently one of multiple crippling arthritis secondary to an infective process in the gall-bladder, and can hardly be regarded as a true case of rheumatoid arthritis.

In Dr. Hale White's case a coccus was found in the synovial membrane of a knee-joint, which could not be identified with any of the more usual cocci. It grew chiefly in pairs, not in chains, was stained by Gram's method, and was not pathogenetic for rabbits. In a mesenteric

gland a small non-motile bacillus was found, which could not be identified. The most recent investigator in this field is Fayerweather. In three cases which he groups as examples of "infectious polyarthritis chronica villosa" he obtained organisms from the synovial fluid and tissues of the affected joints, which were in all instances the only organisms so found. All three micro-organisms were bacilli, but they were all different. Two of the organisms when injected into the joints of rabbits produced arthritis similar in character to that present in the joints from which they were derived. Full description of the organisms, of the cultural properties, and of the animal experiments carried out, as well as of the clinical features of the cases, will be found in Fayerweather's original paper.

Modes of Onset.—There is no uniformity in the mode of onset of rheumatoid arthritis. It may commence as an acute malady, apt to be mistaken for rheumatic fever and sometimes not to be distinguished from it at the onset, which only reveals its true nature by the intractability and persistence of the arthritic lesions. Such errors in diagnosis are sometimes rendered much easier by a history of antecedent attacks, of a transitory character, and separated from each other by intervals of complete or nearly complete recovery. Only after several such premonitory attacks, recurring at short intervals, may the relentless character of the disease ultimately manifest itself. The converse error is also possible, for patients with subacute rheumatism may continue to drag themselves about, and do their daily work, so that their swollen and painful joints, not having a fair chance of recovery, become the seats of such persistent, inflammatory changes that the presence of rheumatoid arthritis is naturally suspected. In such a case a period of rest in bed, with adequate treatment by salicylate, will usually serve to distinguish the class to which the case rightly belongs.

In a larger class of cases the onset of rheumatoid arthritis may be described as subacute, a number of joints are affected in quick succession, but swelling may be insignificant at first, and febrile disturbance minimal or limited to a slight evening rise of temperature. In such cases the slightness or absence of swelling of the joints may prove deceptive, and may lead to a more favourable diagnosis than is borne out by the subsequent course of events. Here, as in acute cases, the implication of joints which are little liable to true rheumatism may prove a valuable aid in distinguishing the nature of the disease.

Lastly, in cases of a more chronic kind, a single joint may be at first attacked, and only gradually does the malady increase its hold and spread to other parts, whilst symptoms of constitutional disturbance may be almost wholly absent. Such attacks, although apparently benign in their earlier stages, are wont to prove the most intractable of all, possibly because the limitation and comparative mildness of the attack do not necessitate rest.

It cannot be too strongly insisted that rheumatoid arthritis, in the sense in which the name is here applied, is not a steadily progressive

malady, but is one which ultimately runs its course, long as that course may be. A sharp distinction must be drawn between the active stage and the sequels, although it is true that these often overlap, so that while some joints may still be undergoing the active changes, other joints of the same patient only shew the havoc wrought by the past arthritis, lesions comparable with those present in the valves of a heart damaged by extinct endocarditis. It is hardly necessary to indicate the bearing of these points upon treatment, for it is clear that the treatment applicable to an acute or subacute inflammatory condition differs widely from that which is called for when we have to deal with joints which are scarred and distorted by thickening of their capsules and muscular contracture.

The shorter the stage of active mischief the less will be the permanent damage produced, and if only it were possible to find means of arresting the disease soon after its onset we should see little deformity and crippling as sequels of rheumatoid arthritis.

The Distribution of the Arthritic Lesions.—The small joints of the hands and feet are the first to be attacked in the majority of cases of rheumatoid arthritis. In the hands the proximal row of interphalangeal joints and the metacarpo-phalangeal joints are specially liable to suffer, whereas the interphalangeal joints of the distal series enjoy comparative immunity from attack in conspicuous contrast to the distribution of the lesions in osteo-arthritis. The wrists too are usually affected early, and with them the knees and ankles. Much stress has been laid upon the symmetry of the distribution of the lesions, which has been advanced as an argument in favour of a nervous origin. Symmetry of lesions, however, is met with in diseases which are undoubtedly infective, such as rheumatic fever, and in this respect the distribution of the subcutaneous rheumatic nodules of young subjects may well challenge comparison with that of the articular lesions of rheumatoid arthritis.

In severe cases almost all the joints may become affected as time goes on, and the lesions shew some tendency to advance from the periphery towards the trunk, the hips being as a rule among the last articulations to be attacked. The liability of the vertebral and temporo-maxillary joints is one of the more characteristic features of rheumatoid arthritis. It is quite unusual for stiffness of the neck and jaw to be absent throughout an attack of any severity, and occasionally the jaw-stiffness is the earliest symptom observed. Nevertheless it is noteworthy that the symptoms referred to the temporo-maxillary joints are usually transitory or intermittent, and in this respect present a contrast to the generality of the articular symptoms. In distinguishing between rheumatic fever and rheumatoid arthritis, the presence of stiffness of the neck and jaw may be of much diagnostic importance, but it must not be forgotten that both the vertebral and temporo-maxillary joints are not infrequently affected in gonorrhoeal arthritis, and are among the seats of election of osteo-arthritis.

It has been thought that in some cases the crico-arytaenoid joints

are seats of the disease, but although in rare cases there may be pain in phonation and tenderness of these articulations, which may very possibly be due to this cause, the occurrence of rheumatoid arthritis in this situation has not, as far as I am aware, been conclusively demonstrated. The same may be said of the joints of the ossicles of the ears.

Clinical Features of the Arthritic Lesions.—As in other diseases which implicate the joints, the symptoms complained of in rheumatoid arthritis are pain, limitation of movement, and swelling. The articular pain varies greatly in intensity in different cases. It will be more conveniently spoken of in a later paragraph together with other kinds of pains which are met with in connexion with this disease.

Swelling of the affected joints is usually a very early symptom, although, as has been mentioned already, it may be wanting in the initial stages, even when the pain is already pronounced. The character of the swelling is somewhat peculiar, for it is largely due to thickening of the capsules of the joints. Hence the enlarged joint has a fusiform appearance, which is in contrast to the more abrupt swelling due to simple synovial effusion, or the lipping and osteophytic outgrowths of osteo-arthritis. The spindle shape is seen in the larger joints, such as the elbows and knees, as well as in those of the fingers, and is emphasised by the conspicuous wasting of the intervening muscles which is so conspicuous a feature of rheumatoid arthritis. In the wrist the swelling abolishes the natural contour of the parts and produces a peculiar humped appearance. Synovial effusion usually contributes to the swelling, and not only the joint capsule itself but also the neighbouring bursae may be more or less distended with fluid. In the later stages the synovial fluid is apt to be viscid and of the consistence of melted jelly, and there are cases in which the accumulation of such fluid is the most conspicuous feature. On palpation it is evident that the swelling is of the soft parts in and around the joints, and in the active stage, now under consideration, no lipping of the bones nor osteophytic outgrowths can be felt. The Röntgen rays confirm the evidence of the fingers, and pictures taken by their means shew the articular surfaces apparently unaltered and the transparent line of cartilage intact. They further shew a peculiar rarefaction of the bones in the neighbourhood of the diseased articulations. Special attention has been called to this feature by Dr. Hale White and others, but as a similar rarefaction has been observed by Kienböck in gonorrhoeal arthritis it cannot be regarded as a diagnostic feature of rheumatoid arthritis.

In some cases a very conspicuous swelling of the joints is due to the distension of the capsule with hypertrophied and arborescent villous outgrowths which fill its cavity. Hypertrophy of the synovial fringes of lesser degree is common in rheumatoid arthritis, but in the cases referred to, of which Max Schüller has made a special study, the hypertrophy is excessive. This condition, which reveals itself during life by the fact that the swollen joints are felt to be filled with soft solid material with irregular surface, may be present in some only of the

affected joints, but is apt to be most pronounced when the disease is limited in extent or even confined to the knees. Whether the villous arthritis of Max Schüller is a variety only of rheumatoid arthritis or a separate disease cannot as yet be definitely stated. The question is an important one in view of Schüller's bacteriological results already referred to.

The axillary and inguinal glands are sometimes swollen, and it is probable that, if always looked for, this glandular enlargement would prove not to be uncommon, even in the case of adult sufferers.

Muscular Atrophy.—Muscular wasting is a common accompaniment of arthritis of any kind, and has been produced in animals by the experimental injection of irritant substances into joints. The degree of atrophy is too considerable to be ascribed to mere disuse, and its distribution in relation to the diseased joints indicates a local cause. It is usually regarded as a reflex atrophy, and is attended by a conspicuous exaggeration of the corresponding tendon reflexes. In rheumatoid arthritis the muscular atrophy is usually so conspicuous that it is held by some to be a primary feature of the disease advancing *pari passu* with the articular lesions, and not a secondary change, but the evidence in favour of this view is not conclusive. In a malady producing such severe articular lesions, which maintain their activity for such long periods, conspicuous wasting of muscles is to be expected, and, further, from the large number of joints so often affected, the atrophy is likely to be unusually widespread and conspicuous. Although often so extreme in degree the wasting has the ordinary characters of arthritic atrophy. The tendon-jerks are found to be increased when the state of the joints allows them to be elicited, and only in extreme and long-standing cases is the reaction of degeneration to be obtained in any of the muscles. On the other hand, there are some puzzling features. The amount of atrophy is singularly unequal in cases which appear to be of equal severity; whereas in one case the long bones appear to be almost devoid of muscular covering, the spindle-shaped swelling of the joints being thereby greatly emphasised, in another the nutrition of the muscles is fairly well maintained throughout the illness. The view which regards the atrophy as primary gains most support from a peculiarly malign but fortunately rare variety of the disease, in which the distortions and deformities due to muscular contractions appear almost at the onset, whereas the swelling of the joints is comparatively insignificant, and the articular lesions appear hardly sufficient to account for the muscular changes. In these cases a degree of crippling is reached in the course of a few months, which in cases of the more ordinary kind would only be produced in the course of years.

The Joint Lesions in the Stage of Sequels.—When the active stage of rheumatoid arthritis has passed, either in individual joints or in the joints at large, pain diminishes greatly and swelling subsides, but it is only in exceptional cases, in which the active stage has been unusually short, that the contour of the affected parts is fully restored. Some

thickening of the capsule almost always remains. The muscles which have remained in a state of atrophy over long periods also fail to recover, and undergo contracture and permanent shortening. These changes lead to more or less fixation and displacement of the articular surfaces upon each other. Complete extension of the knees may become impossible, and there may be almost complete inability to walk. In the hands the deformities from contracture are particularly well seen, and the tension of the tendons is apt to produce permanent flexion of some of the joints and hyperextension in others. The deformities which result were elaborately classified by Charcot under two main varieties and a number of sub-varieties. Into the details of this classification it is unnecessary to enter here, and it may suffice to state that in the flexed variety the terminal phalanges are hyperextended upon those of the second row, and these again are flexed upon the proximal phalanges. The first phalanges are also flexed upon the metacarpals and the carpus upon the forearm. In the extension form, on the other hand, the interphalangeal joints of the proximal row are hyperextended, whereas those of the distal row are flexed. The above deformities are exactly similar to those seen in the hands of patients distorted by muscular contracture apart from any lesions of joints.

Ulnar deflexion of the fingers, on the other hand, which is a common sequel of rheumatoid arthritis, appears to be intimately connected with damage to the metacarpo-phalangeal joints, and is not seen in cases of contracture apart from articular lesions. It is in no way peculiar to the disease under discussion, although most frequently seen in its victims. The exact mechanism of its production is still doubtful. A further result of the contracture is subluxation of joints, which is not uncommon in the knees from the tension of the shrunken hamstring muscles, and is also frequently seen in the metacarpo-phalangeal joints. These deformities are permanent, and, in cases of long-standing, complete immobility and true bony ankylosis may result. When the muscular atrophy is comparatively slight the prospects as regards crippling are much more favourable. The joints, although left somewhat thickened and enlarged, may recover their mobility to a great extent, so as to allow of such fine movements as are involved in sewing or writing. The muscles too may regain their original contour.

The joints themselves may be left so seriously damaged that the incapacity of the patients is seriously increased thereby. In severe cases of long standing post-mortem examination may shew the cartilages to be completely destroyed, and during life there may sometimes be felt, especially in the knees, a peculiar scraping as of one bare bony surface upon another. Bony excrescences may develop around the articular surfaces, but, unlike the ordinary osteophytes of osteo-arthritis, these are rather bony spicules which are often peculiarly sharp-pointed.

Pain.—The articular pain, which is directly attributable to the inflammatory changes in the joints, is of very unequal intensity in different cases, and its severity affords no aid in prognosis, whereas in

some cases of a comparatively mild kind, such pain is one of the most conspicuous features; in others which are much more severe it is almost insignificant. Pain may be spontaneous and present when the joints are at complete rest, but is naturally aggravated by movement of the affected parts. It is often markedly increased by the warmth of bed. It has none of the shifting character of the pains of rheumatic fever, and extension of the disease is attended by increase of the area of pain. A second variety of pain, which is a conspicuous and distressing feature in not a few cases during the more advanced stages of the disease, is due to spasmodic contraction of the atrophied muscles. This muscular cramp is usually confined to a limb or to part of a limb, but the patient's sufferings are apt to be aggravated by the tension and pressure exerted upon inflamed and tender articular structures. Yet a third kind of pain, which may attain to great intensity, is associated with arthritis of the vertebral joints. It is a true causalgia following the course of the intercostal nerves or taking the form of brachial neuralgia, and is to be ascribed to implication of the nerve-roots as they emerge from the spinal foramina. Definite nerve-root tenderness may usually be elicited in the affected area. It is also probable that pain following the track of nerves results from a primary neuritis.

Subcutaneous Nodules.—Hard masses can sometimes be felt in the olecranon bursae, and sometimes in the soft structures at a distance from any joint. In addition, small nodules, closely resembling the subcutaneous nodules of true rheumatism, are sometimes seen in the neighbourhood of the inflamed joints. Stress has been laid by some upon the occurrence of such nodules as affording evidence of a common origin of rheumatoid arthritis and rheumatism; but the nodules of rheumatoid arthritis, which are much more permanent than the rheumatic ones, and, unlike them, sometimes extremely tender, are for the most part met with in patients who have reached ages at which true rheumatic nodules are unknown. The latter are essentially manifestations of rheumatism in childhood, and though sometimes seen in early adult life, their age incidence is practically the same as that of chorea; further, they are almost always associated with conspicuous cardiac damage.

Cutaneous changes are present in many cases of rheumatoid arthritis, in the form of trophic lesions and abnormal pigmentation. In cases of a malign character the surface of the skin is apt to appear greasy, the hands and other parts may be cold and moist, and the skin of the fingers may be atrophic, pink, and glossy. Pigmentation, which is often seen, and is sometimes a very conspicuous feature of the disease, was first described by Dr. Kent Spender, from whose graphic description (this *System*, 1st ed. 1897, vol. iii. p. 85) the following passage is quoted:—"Concentrated in patches more or less large, the pigment assumes many hues and affects many parts of the body. Across the forehead it spreads as a light bronze smear, or like a dash of chloasma; over the temporal fossa on each side the tint may be deeper. Under the lower eyelids the

streak of colour may be very dark, and may shine with a metallic polish. The dominating tints on the face are lemon, orange, and citron; the lustre varies with the angle of reflected light. In people of dark complexion the colour of the face is sometimes so swarthy as to make the patient resemble a mulatto. The white of the eye stands out in brilliant contrast. In some cases the neck looks as if soaked in walnut dye, beginning at the line at which it is ordinarily covered, and the skin has the appearance of being unwashed." In addition to the diffuse and smeary pigmentation here described, sharply circumscribed freckles are also present, as Dr. Spender also observed, and these may vary in colour from a light brown to black. They are often seen upon the backs of the hands, on the forearms, forehead, and indeed in almost any situation. The pigmentation is a phenomenon of the active stage of the disease, and as the patient's general health improves and the active trouble subsides, the skin tends to resume its ordinary appearance.

Oedema of the feet and legs is sometimes present in very severe cases, and especially in crippled patients spending many hours of the day in a sitting posture. Such oedema is quite independent of lesions of the heart or kidneys. It is usually of the brawny kind, and the parts hardly pit at all on pressure. Ulceration of a most intractable kind sometimes occurs upon the overhanging brawny folds.

Constitutional Symptoms. — The febrile disturbance of rheumatoid arthritis is very variable. In some severe cases there is hardly any elevation of temperature, in most there are evening rises and morning remissions. The maxima are not infrequently as high as 102° or 103° F., and Dr. Bannatyne has seen as high a reading as 105°. As Kahler pointed out, periods of high temperature may recur at intervals, separated from each other by comparatively afebrile intervals. The patient is usually but little conscious of the febrile disturbance, which plays a comparatively insignificant part in this disease. In febrile cases return of the temperature to the normal throughout the twenty-four hours is a valuable sign of the completion of the active stage of the disease, but the absence of fever throughout is consistent with conspicuous activity of the morbid process.

Some degree of circulatory disturbance is another symptom of rheumatoid arthritis, the recognition of the importance of which we owe to the clinical acumen of Dr. Kent Spender, whose article may again be quoted in this connexion: "The changes in the circulation are frequent and important. Many cases of rheumatoid arthritis, rapid and crippling in their march, are characterised almost from the beginning by increased rapidity of the heart's action. The pulse-rate may go up gradually to 90 or 100, and remain so for years. This is tolerably common. In some comparatively rare but very noteworthy cases the pulse may run higher still. Synchronously with the earliest objective signs of arthritic disease the cardiac rate may rise to 110, 115, or 120. Scarcely any variation is observed, whether by day or night. I have recorded one case in which a very hard pulse reached the high point of 140." Clinical experience

confirms the accuracy of these statements. The rapidity of the pulse has no obvious relation to the febrile disturbance. It is observed alike in pyrexial and apyrexial cases, and long after the more active stage of the malady is past the rapid action of the heart may persist, the rate returning only very gradually to the normal limits. From the standpoints of both diagnosis and prognosis the rapid pulse of rheumatoid arthritis is of considerable value and importance.

Pallor and cachexia are prominent in many severe cases, and this form of secondary anaemia may also be classed as a symptom of the active stage. As this subsides the general improvement of health is attended by improved condition of the blood, but in crippling cases the enforced sedentary life and confinement are apt to be attended by some lasting anaemia.

Depression of spirits can hardly be avoided in sufferers from such a prolonged and painful disease, which is apt to be followed by permanent disability, and to cut the sufferer off from most of the enjoyments and many of the interests which make life worth having.

Visceral Manifestations.—Although rheumatoid arthritis has all the characters of a systemic disease its local manifestations are almost confined to the joints, muscles, and superficial structures. Visceral lesions are rare, and when they occur are usually to be ascribed to some intercurrent malady. In the very great majority of cases rheumatoid arthritis runs its course without any signs indicative of inflammatory changes in the endocardium or pericardium, and without the acute dilatation of the heart which is a sign of myocarditis. When an organic valvular murmur appears in the course of this disease it comes as a surprise rather than as a looked-for event. In the present state of our knowledge, however, it must not be assumed that the cases in which such lesions develop belong to the rheumatic rather than to the rheumatoid group; the possibility that rheumatoid arthritis does sometimes though rarely attack the heart cannot be denied, nor, if we are right in regarding it as an infective malady, is this surprising, seeing that the endocardium has been shewn to be liable to infection by numerous bacteria, including the ordinary pyogenetic cocci, the gonococcus, the pneumococcus, and the bacilli of influenza and of tuberculosis. The presence of a murmur is not of much importance, unless it has been observed to appear during the illness, for even the absence of a history of rheumatic fever by no means excludes a remote rheumatic origin. Dr. Bannatyne's large experience leads him to look upon cardiac complications as much more common than most writers allow. In a series of 293 cases cardiac lesions appeared in the course of rheumatoid arthritis in 17·9 per cent, and he puts the proportion of cases with pericarditis at 4 or 5 per cent. I have never seen active pericarditis in a case of rheumatoid arthritis, but I have found pericardial adhesions after death in a case in which the active stage of the disease was long past, and in which no history of acute pericarditis was forthcoming.

Nephritis with the passage of urine loaded with albumin may be

mentioned as an occasional but rare event in cases of the gravest character.

Scleritis, iritis, and conjunctivitis have been described as complications of rheumatoid arthritis, but it is doubtful whether they are really manifestations of this disease. Their occurrence would suggest that a gonococcal infection is responsible for both the arthritic and the ocular lesions.

Rheumatoid Arthritis in Children, and Conditions which Simulate it.—The question whether or no rheumatoid arthritis occurs in children, and if so, what proportion of the cases in which persistent lesions of joints are seen in early life are really of this nature, is beset with difficulties, and calls for special consideration.

The more chronic arthritides of children are of several distinct kinds; this was clearly pointed out by Dr. G. F. Still in his articles on this subject, which have justly met with a wide recognition. Some rare cases, which in their clinical aspects recall the osteo-arthritis of advanced life, present lipping of bones and nodes like those described by Heberden. Of the morbid anatomy of such cases we are almost wholly ignorant, and their pathology is obscure, but probably, as Dr. Still suggests, congenital syphilis is a factor in the etiology of some of these cases. Nor must it be forgotten, as Mr. Bowlby shewed, that osteo-arthritic changes are met with in joints which have been the seats of the arthritis of haemophilia. In another class of cases, which are also rare, chronic articular lesions result from attacks of true rheumatism. These lesions which are characterised by fibrous thickening of the periarticular structures, may result in fixation and deformities recalling those seen in the stage of sequels of the rheumatoid arthritis of adults. Yet in their course and clinical features these rare cases present special features which mark them out as belonging to a distinct class, and may be grouped with the condition met with in adults which has been described by Jaccoud as *rhumatisme fibreux*. There remains a larger group of cases in children with fusiform swelling of joints which present a clinical picture resembling more or less closely that of rheumatoid arthritis as seen in adults. It is almost certain that this group includes some cases of rheumatoid arthritis occurring at an unusually early age, since rheumatoid arthritis may occur at any period of adult life; in quite young women it is by no means rare, and no period of immunity can be traced breaking the chain of incidence between childhood and maturity. Nevertheless there are arguments of some cogency which may be adduced in support of the view that the bulk of the cases of articular disease in childhood presenting the rheumatoid features are in reality not of this nature, but are rather examples of a distinct malady, which at the present day is often spoken of as "Still's disease."

The features clinical and anatomical of such cases are well given in the following description, which is quoted at length from the article contributed by Dr. Still to the first edition of this work (1897, vol. iii. p. 103):—

"The disease mentioned first may be defined as a chronic progressive enlargement of joints, associated with enlargement of lymphatic glands and spleen.

"Its *causes* are unknown. Heredity seems to play no part in its causation. Bad feeding, privations, and faults of hygiene were by no means prominent in the cases observed.

"The onset is almost always before the second dentition: ten out of twelve cases began before the age of six years, and eight of these began within the first three years of life; the earliest was at fifteen months. Girls are somewhat more liable to be affected than boys; seven of the twelve cases were girls, five were boys.

"The *morbid anatomy* is revealed by three autopsies which were made at the Hospital for Sick Children. In two cases, which had lasted fourteen months, the joints, which were markedly enlarged, shewed only increased vascularity of synovial membrane and ligaments, with slight increase of synovial fluid and thickening of the capsule, which, in one case, was very obvious. In a very advanced case of three years' standing there was, in addition to thickening and increased vascularity of the synovial membrane, some pitting of the cartilage at its margin, giving it in some parts a worm-eaten appearance; little processes of the thickened synovial membrane fitted accurately into these pits, and thus the changes in the cartilage seemed to be secondary to the changes in the synovial membrane; the rest of the cartilage was healthy. There were some fibrous adhesions in the joints in this case. The fibrillation of the cartilage, which is so characteristic of rheumatoid arthritis, even in its early stages, was absent in these cases; osteophytic change, and exposure and eburnation of bone, were also absent. The glands and spleen shewed considerable enlargement, but on section appeared normal, save for a few small ecchymoses which were seen in the substance of the glands. The pathology of this disease is uncertain; but some of the symptoms are at least suggestive of a microbic origin.

"*Symptoms.*—The onset is usually insidious, with stiffness of one or more joints, which slowly become enlarged; but occasionally the onset is acute with pyrexia and, it may be, with rigors. The character of the joint enlargement is almost constant. It suggests rather thickening of the tissues round the joint than a bony enlargement. There is none of the bony irregularity of rheumatoid arthritis, and the absence of bony thickening and lipping, even after the disease is considerably advanced, is striking. Bony grating cannot be obtained, but creaking of tendon or cartilage is frequently present. Effusion is seldom a marked feature. Tenderness if present is usually slight. There is generally some limitation of movement. The child whose photograph is shewn in Fig. 1 was completely bedridden at the age of four years, owing to an almost rigid flexion of all the larger joints. The joints earliest affected are the knees, wrists, and cervical spine. The fingers soon become affected, and later the toes. The sterno-clavicular and temporo-maxillary joints are but rarely implicated. The disease is probably never limited to one joint:

it is almost always symmetrical. There is no tendency to suppuration in the joints, nor to bony ankylosis.

"The muscles of the limbs waste considerably as the disease progresses; they may undergo contracture; thus the hamstrings became shortened in a case where the knee was kept constantly flexed. The electrical reactions are unaltered.



FIG. 1.—Chronic arthritis with enlargement of lymphatic glands and spleen in a girl aged four years.

"Enlargement of the lymphatic glands is a constant symptom. It affects primarily and chiefly those related to the affected joints, but may become more general. The glands are separate, rather hard than soft, not tender, and shew no tendency to break down. They may become so large as to be visible, but more often do not become larger than a hazel-nut. Their size varies with the progress of the joint affection; improvement in the joints is followed by diminution in the size of the glands.

"Enlargement of the spleen is almost always detected. It was found in nine out of twelve cases. The edge of the spleen is felt 1 to 1½ inch below the costal margin. The enlargement is roughly proportionate to that of the glands, and varies with the joint condition.

"The heart shews no evidence of valvular disease, but haemic bruits are sometimes present. A tendency to inflammation of pleura and pericardium seems to exist in these cases: in three cases this was shewn post-mortem by old adhesions, and in two other cases there were physical signs suggestive of adherent pericardium. Moderate anaemia is generally present. Sweating is often profuse. A curious occasional symptom is slight prominence of the eyes; it is not associated with any enlargement of the thyroid gland. In cases beginning before the second dentition there is often a marked arrest of bodily

development; a child of twelve and a half years of age, in whom the disease began at four years of age, still had the appearance of a child of six or seven. The temperature in some cases is almost continuously raised to 100° or 101° F.; in others short periods of pyrexia alternate with longer periods of apyrexia.

"The course of these cases is almost always slow. Temporary improvement is common; but, so far as I am aware, no recovery has been recorded: the child in the course of months or years becomes an almost helpless cripple. The disease is not in itself fatal. Three deaths have occurred at the Hospital for Sick Children, two at the age of three and a half years, one at four years, due respectively to bronchitis, erysipelas, and pneumonia. A curious result of certain accidental complications was observed; in one case catarrhal jaundice, in another scarlet fever, in a third measles, were followed by definite improvement in the joint condition.

"*Diagnosis* in the early stage is often a matter of extreme difficulty. Where the onset is acute the disease is generally mistaken for acute rheumatism, until the persistence of the joint affection and the enlargement of the glands and spleen reveal its nature. Where the onset is insidious the early affection of the spine may suggest spinal caries; and I have more than once known the difficulty in walking which was due to the early affection of the knees to be attributed to rickets. The symmetry of the joint affection, the large number of joints involved, and the absence of any tendency to caseation, will generally suffice to exclude tuberculous disease. The joint lesion of congenital syphilis may closely simulate this disease; but usually the history and the presence of other symptoms of the inherited infection will distinguish them. The diagnosis from the disease known as rheumatoid arthritis in adults, and from chronic fibrous rheumatism, is often very difficult in the early stage."

The accuracy of the above description will not be questioned by any one who has experience of such conditions. It will be noticed that the morbid appearances found in the joints after death resemble in almost all respects those met with in cases of acute and subacute rheumatoid arthritis in adult subjects, and if the distinction drawn in the present article between rheumatoid arthritis and osteo-arthritis be a true one, all arguments (p. 18) based upon the absence of the osteo-arthritic changes from the joints of such children lose their force. It is on the clinical side that the distinguishing features are seen, and the wide differences in the clinical features of true rheumatism as seen in adults and in children respectively bid us pause in accepting too readily distinctions based on clinical grounds alone. Undoubtedly, enlargement of lymphatic glands is a far more constant and conspicuous event in the children's disease than in the rheumatoid arthritis of adults, and the same may be said of splenic enlargement, although in my experience this cannot be made out quite so frequently as Dr. Still's original cases indicated. It must be remembered, however, that the splenic enlargement may only be

recognisable during the more active stages, and may be missed during the quiescent periods.

In not a few cases the arthritis is limited to the larger joints, such as the wrists, elbows, knees, and ankles, and the digital and other smaller joints escape, and again the temporo-maxillary joints are far less prone to be attacked than in the adult cases.

Of recent years the suggestion has come from several quarters that some at least of the cases in question are tuberculous in their nature; not that the affected joints are the seats of gross tuberculous lesions, but that the disease is a form of the *rhumatisme tuberculeux* of Poncet (see p. 57). Mouriquand has described a case, with enlargement of lymphatic glands, to which he ascribes a tuberculous origin, although the evidence adduced is not very convincing. Edsall records the case of a boy, aged thirteen, who presented all the characteristic features of Still's disease, who repeatedly reacted to injections of tuberculin, with intense pain and tenderness in the affected joints during the reaction. In some lymphatic glands removed from the axilla no bacilli were seen in sections, but in an emulsion prepared from these glands many acid-fast bacilli were found, indistinguishable from tubercle bacilli. But as the injection of the emulsion into guinea-pigs produced no tuberculous lesions, Edsall concludes that they were probably dead, or of very low virulence. Dr. Parkes Weber inclines to the view that some cases at least are probably tuberculous, and describes a case in which a positive reaction to tuberculin was obtained, but no local reaction was noticed in the joints.

In conclusion, it is evident that our present knowledge does not suffice for the decision of the question of the relation of the disease met with in children to rheumatoid arthritis, and it is probable that the final solution of the problem will come from the bacteriological side.

Diagnosis.—It is a question whether rheumatoid arthritis, as here described, is a distinct disease, and not rather a medley of infective arthritides of various kinds. As far as uniformity of clinical features can be relied upon as a guide, there would seem to be little room for doubt that such a specific disease does exist, and that the majority of cases of rheumatoid arthritis are of one definite kind. On the other hand, it cannot be denied that a number of infections may bring about a condition of the joints which may simulate very closely the rheumatoid lesions, and it is a matter of great difficulty to decide what proportion of such extraneous cases are included even by such observers as are not content to classify under this head any forms of arthritis which run a prolonged course, are little amenable to treatment, and do not fall into any other recognised group. For example, it is doubtful how far the existence of a definite infective focus, such a pyorrhoea alveolaris, is to be taken as affording evidence that a case is not one of true rheumatoid arthritis, and we are hardly justified in assigning to the disease under consideration so malignant a character that an early recovery, without permanent impairment of function, excludes the diagnosis. It may be that, ere long, a more extensive study of the bacteriological aspects of the problem will supply

us with a definite criterion, by which the diagnosis of rheumatoid arthritis may be established or negatived in any particular case, or may even shew that there is no such specific malady. There is little chance that such a degree of precision can be reached on clinical lines alone. Our knowledge of the joint lesions due to some of the well-recognised bacteria, pyogenetic and other, is still very far from complete, and only in recent years has an arthritis, sometimes met with in association with pneumonia and sometimes apart from any such lesion, been shewn to be due to the presence of the pneumococcus in the articular structures.

The diagnosis of rheumatoid arthritis from true rheumatism is by no means always easy. At the onset of an acute attack of the former disease it may be wellnigh impossible, especially when it has been preceded by transitory attacks of pain and stiffness in joints. The chief aids to diagnosis are the persistence of the articular swellings, the scanty effects of anti-rheumatic drugs, and the implication of such joints as the temporo-maxillary, which are little or not at all liable to be attacked in rheumatic fever. The difficulty is further increased because rheumatic fever occasionally, though rarely, leaves in its wake persistent swelling of joints, giving them a fusiform appearance, which closely resembles the effects of rheumatoid arthritis. More rarely still fibrous thickening and muscular wasting serve to bring about a condition of crippling and deformity in truly rheumatic cases. Whilst it can hardly be doubted that some rheumatic cases of these latter kinds are diagnosed as examples of rheumatoid arthritis, there do not appear to be any sufficient grounds for the belief, held by some, that there is an intimate causal relationship between the two maladies, in other words, that rheumatoid arthritis is a form of rheumatism. In the great majority of instances rheumatoid arthritis shews its distinctive characters from the first, no rheumatic antecedents are to be traced, and none of the visceral manifestations of true rheumatism appear. The liability of the heart to be affected is little if at all greater than in gonorrhoeal rheumatism. The fusiform swelling of joints sometimes persisting after rheumatic fever, more especially in cases of a very subacute kind, in which the pain and stiffness are not sufficient to impose rest, may be indistinguishable from that produced by rheumatoid arthritis; but after persisting for a time, and exciting grave apprehensions as to the future course of the case, the swelling tends to subside and complete recovery usually results.

Gonorrhoeal rheumatism, also, is sometimes followed by a condition indistinguishable from rheumatoid arthritis, in which joint after joint becomes attacked, the active stage continuing over a long period. Such cases must be distinguished from those in which fibrous adhesions and muscular wasting bring about complete fixation of joints and crippling such as results from fibrous rheumatism. If in the cases of the rheumatoid class the progressive disease is really due to the gonococcus and not to a secondary infection, we are bound to suppose that there is a lingering gonorrhoeal infection of the deeper urinary passages, for crippling disease may be separated by a long interval from the primary gonorrhoeal

rheumatism. In two very remarkable examples which came under my notice, attacks of gonorrhoeal rheumatism in the knees were in each instance followed by intermittent hydrarthrosis of those joints (*vide* p. 60). The hydrarthrosis recurred at periodic intervals, so regularly that the patients could predict the condition of their knees at any particular date; and after this periodic affection had persisted for years, without any benefit from treatment, there commenced a general arthritis with fusiform enlargement of the joints, the intermittent hydrarthrosis ceased, and the patients, hardly any of whose joints ultimately escaped, both became completely crippled.

The diagnosis of rheumatoid arthritis from gout seldom offers any special difficulty, but there are cases in which gout attacks large numbers of joints and runs an unusually long and asthenic course, in which such confusion is possible. In such cases the diagnosis is usually cleared up by the previous history of the patient, the course of the disease, and the effects of appropriate treatment.

As regards the distinction of rheumatoid arthritis from the less-known forms of infective lesions of joints no definite indications can be given. In some cases the limitation of the disease to very few joints, and those of the larger kind, will excite suspicion, for the tendency to wide extension is one of the characteristic features of the malady. In others treatment of a local infective focus is promptly followed by an amelioration, and in a short time by complete recovery of the affected joints. Yet it is necessary to be careful lest in directing our attention too closely to the particular features of individual cases we lose sight of those features which they share in common.

Prognosis.—Of diseases which do not directly threaten life there are few if any in which the outlook is so forbidding as with rheumatoid arthritis. We are painfully conscious of the little power which we possess to control it by treatment, and the relentless character of the malady is constantly being brought home to us. Nevertheless in their course, and in the amount of crippling which they leave behind them, individual cases differ widely.

In any given instance two distinct elements in prognosis must be considered, namely, the prospects of arrest of the active disease and the degree of deformity which is likely to follow in its train. An adequate opinion on the first point can only be arrived at by watching the course of the case, and especially the constitutional symptoms. In acute cases a gradual return of temperature to the normal limits, and a gradual diminution of the pulse-rate, together with diminishing pain and swelling, are, naturally, most encouraging signs; whereas in cases running a more chronic course, the conditions of the joints is chiefly to be relied upon. It is by no means the most acute attacks which tend to run the longest course. Of no less importance is the cessation of that tendency to attack fresh joints which is so conspicuous a feature in the more active stages of rheumatoid arthritis, both as indicating arrest and as limiting the range of the damage done.

As regards the ultimate prognosis the degree of the muscular atrophy is, perhaps, the chief factor to be considered. When atrophy is extreme the resulting contracture-deformities are likely to be most pronounced, and in those rare examples in which the contracture-deformities begin to develop in the earliest stages, when the swelling of joints is comparatively slight, the outlook, as regards crippling, is especially gloomy. The damage done to the joints, and especially hypertrophy of synovial fringes, may in itself lead to serious incapacity apart from muscular contracture.

Speaking generally, the shorter the active stage, the better is the ultimate prognosis, and in cases in which the inflammatory lesions quickly subside, the joints, although they usually shew some permanent enlargement, due to thickening of their capsules, may retain their mobility, and there may be little or no crippling. It must not be forgotten that the disease is liable to relapse, and even in the most favourable cases the course of recovery is seldom unbroken. Patients whose original attack has subsided quickly and has left them comparatively unscathed may become completely crippled as the result of a relapse.

Death is usually due in the end to some intercurrent disorder, to which the patient more easily falls a victim in consequence of the enforced inactivity, and the depression of spirits which the malady can hardly fail to bring about. Occasionally the patient appears to succumb to the disease itself during the active stage, becoming more and more anaemic and failing in all respects, but this is certainly a very rare event, and is seen in the most malignant cases only.

Treatment of Rheumatoid Arthritis.—The distinction, so repeatedly emphasised already, between the active stage of rheumatoid arthritis and the stage of sequels, has nowhere so much importance as in connexion with treatment. In order that any real progress may be made in this direction it is essential that it should be recognised that in the early stages we are dealing with inflamed joints, and not with joints which are the seats of a mere degenerative process.

Rest.—As in other diseases in which arthritis occurs, the inflamed articulations should be subjected to no violence, but should be guarded and kept at rest. Yet it is difficult to attain this end, seeing that patients are usually imbued with the notion that the joints should be kept in movement with a view to the avoidance of subsequent stiffening. It is probable that during the acute stage rest is the most important of all therapeutic measures in rheumatoid arthritis. This view is supported by the fact that the active stage is often comparatively short in acute cases, in which the state of the joints imposes rest almost from the first, and is wont to be most prolonged in those in which the sufferers are at no time so incapacitated that they are unable to drag themselves about. In such circumstances the disease is apt to smoulder on for many years, never very active in many joints at the same time, but gradually attacking fresh articulations, whilst those first affected are passing into the stage of crippling. Could complete rest be imposed in every case at the outset we may well believe that there would be far

fewer victims of those extreme forms of crippling which rheumatoid arthritis so often produces.

Massage.—With a view to counteracting muscular atrophy, systematic massage of muscles, carried out once or twice a week, for months together, is probably of real value in averting the more grave forms of contracture deformity. The massage must be adapted to the condition of the patient, and when gently and skilfully done can usually be practised throughout the acute stage, provided, as should always be firmly enjoined, the joints themselves are left severely alone. If the patient cannot tolerate the massage it should be postponed to a later period. It is hardly necessary to point out how great is the difficulty of getting such treatment carried out. Patients are naturally disinclined to persevere with therapeutic measures which are attended by no immediate and obvious benefit, and which are merely measures of insurance against contingencies, the risk and importance of which they cannot be expected to realise. When it is carried out, the results obtained and the reduction to a comparatively limited extent of the contracture-deformities may be very striking.

Diet.—It is generally agreed, at least among those who have taken any special interest in this disease, that nothing is gained, whereas much may be lost, by restrictions in the dietary of the patients other than those which may be required by the constitutional condition. There is no evidence that any special articles of diet have a deleterious influence in these cases, although it is often by no means easy to combat the widespread notion that a diet such as is believed to be beneficial to gouty subjects is necessary for all whose joints are the seats of disease, whatever the nature of the disease may be. Our aim should be to bring our patients into the most favourable state of nutrition that is possible, and so to ensure to them every advantage in combating the disease; since, as a result of confinement, depressing conditions, and continuous pain, the appetite often tends to fail, and we are more often called upon to press a more generous diet than to impose restrictions. Proteins may be freely given, in forms suited to the febrile or afebrile condition of the patients. Fats, such as cream and cod-liver oil, are often of much service, and carbohydrates may be given as digestion allows. Only when the patients tend to lay on fat to an undue extent, which is seldom or never the case before the active stage of the disease is over, is it necessary to limit materially the intake of the more fattening foods, since increasing corpulence brings in its train increasing incapacity. Extreme plans of dieting, such as placing the patients upon an almost wholly protein diet for a time, are credited with conspicuous success in a few cases, but in the majority of cases of rheumatoid arthritis they appear to do more harm than good. There is no evidence that alcohol has any share in the causation of the disease, nor that alcoholic beverages are specially harmful to those who suffer from it. Nor are any special indications present for the taking of spirits rather than wines or malt liquor. It would seem, however, that in some cases

aching joints are rendered more painful by the taking of alcohol, irrespective of the nature of their disease. Whether we recommend the use of alcohol in this malady will depend rather upon the views which we hold on the wider question of its therapeutic uses in general.

Climate.—A warm dry climate and a dry soil are the most suitable to patients with rheumatoid arthritis, and the climate selected should be as equable as possible. If the patient reside in a damp and low-lying neighbourhood, removal to a drier locality should be urged. In choosing a residence, however, it is advisable that a trial be made of the locality proposed before a permanent home is set up. Well-to-do sufferers often obtain benefit from wintering in a warmer climate than our islands afford, and in selecting a winter resort the desirability of an equable warmth, such as can hardly be obtained on this side of the Mediterranean, should be considered. The climate of Egypt suits many such patients well, and the same may be said of that of the Canary Islands, although those who remain at home usually find that proximity to the sea brings with it increase of pain. In advising for or against wintering at a distance the condition of the patient has to be taken into serious consideration. When there is much crippling the exhaustion and discomfort which a long journey entails may more than counterbalance the advantage gained, and there are not a few sufferers whom one is reluctantly driven to advise to stay at home on such grounds.

Spa Treatment.—The victims of rheumatoid arthritis form a large proportion of the patients at many spas, and the treatment there carried out is often of great benefit. Patients are, however, sometimes sent for treatment at stages in which such courses are apt to be injurious rather than beneficial. It is a good rule to avoid sending those to whom one would certainly not recommend such treatment if they were suffering from any other form of infective arthritis, and in whom the joints are the seats of acute or subacute inflammation in an aggressive and spreading stage. Later on, when the active stage has subsided and when the damaged structures threaten to become permanently deformed, balneo-therapy may prove of great service. In the more acute stages simple immersion baths are of course not open to the same objections as such active measures as douches and massage, but it is probable that complete rest is preferable to any bathing. The spas which enjoy a reputation for the successful treatment of the results of this disease do not belong to any one class, but differ widely in the composition of their waters. The mode of application of the waters is of more importance than their chemical properties, and the accessory factor of climate appears to have no slight influence in determining the amount of benefit received. The combination of douche with massage, which had its origin at Aix-les-Bains, appears to be decidedly useful in many cases in which the acute stage is past. The Aix treatment is now carried out at several British spas, and notably at Bath, where alone in our islands a supply of water raised by nature to a high temperature is available. Harrogate, Buxton, and Woodhall Spa may also be mentioned among those British

watering-places at which sufferers from rheumatoid arthritis often obtain relief. Brine baths, such as those of Droitwich and Nantwich, prove useful in some cases in relieving stiffness and deformities.

Peat baths, such as those at Strathpeffer and many other spas, are sometimes of service in relieving pain, and if a conspicuous degree of anaemia is a feature of the case, resort to iron springs, such as those of Schwalbach, Spa, or St. Moritz, may be desirable.

Vapour-baths almost always give much relief for the moment, and by reducing pain and stiffness frequently improve the patient's powers of locomotion, but frequent baths of this kind are not to be recommended. This does not apply to the local vapour-baths of the Berthollet type, in which steam is applied to the neighbourhood of the joints only.

Electric baths should also be mentioned among forms of treatment which have proved useful in many cases, and pain and stiffness of joints are also relieved by the application of hot air, whether the heat be derived from electric lamps, or whether the air be heated in other ways. Such treatment also has most chance of success in the earlier days of the stage of sequels.

Treatment by drugs is not so efficacious as might be wished, and no drug has as yet established a claim to any such efficiency as the salicylates possess in relation to rheumatic fever. Of the many remedies which have been recommended guaiacol carbonate, first advocated by Dr. Bannatyne, given in gradually increased doses and for long periods, for months or for a year, has the strongest claims, and no case in which this drug can be tolerated, as it almost always can, should be left without a trial of this plan of treatment. In not a few cases such treatment gives very encouraging results, and the active stage of the disease appears to be materially shortened. This is more especially the case when an iodide is given at the same time, as Dr. Luff recommends, and iodide of iron is often advantageously employed. It is, however, in early and mild cases of osteo-arthritis rather than in those of rheumatoid arthritis that iodide of iron appears to be specially useful. Only by the prolonged administration of these drugs are favourable results obtained.

Salicylates often relieve the articular pains, although they do not appear to exert any specific action in rheumatoid arthritis. Aspirin is the most efficient for this purpose, and gives immense relief in some cases, but in rheumatoid arthritis, as in other painful conditions, it may fail entirely, nor can it yet be confidently asserted that when given continuously over long periods it is innocuous.

As local applications guaiacol, methyl salicylate, or linimentum potassii iodidi cum sapone sometimes prove useful, or a liniment of belladonna and iodine.

Rheumatoid arthritis is among the morbid conditions in which benefit may be obtained by the induction of hyperaemia, a plan of treatment very extensively employed, especially upon the Continent, and closely associated with the name of August Bier. The

hyperaemia induced for therapeutic purposes may be either active or passive, and in this country the active form is mainly employed, the hyperaemia being induced by hot-air baths of the Tallerman kind, or by radiant-heat baths, in which electric lamps are the sources of heat. Bier also recommends the induction of passive hyperaemia by means of elastic bandages. This plan is of most service for the treatment of the more peripheral joints, whereas for such joints as the shoulders or knees hot-air baths are to be preferred. A thin Martin's bandage is applied to the limb above the joint to be treated, the successive turns overlapping, so that a considerable area is bandaged. The tightness of the bandage should be so adjusted that the arterial circulation is not arrested, whereas the venous circulation in the limb is to a great extent arrested. A bandage so adjusted may be borne for hours without any serious inconvenience, although the skin assumes a bluish-red tint, and oedema results if the pressure is kept up for a sufficient time. Bier recommends the application of the bandage in these cases for as long as 10 to 22 hours per diem, the oedema which results being removed by one or two applications of massage daily, the massage consisting in a stroking of the limb from the periphery upwards. Unless the bandage be applied sufficiently tightly to produce an obvious oedema of the limb it will have little effect. The treatment must be persisted with for a considerable time, but after it has been continued for a few weeks or months, an interval of one to four weeks should be allowed. Bier considers that the results of such treatment compare well with those obtained by any other means, but warns against the entertaining of too high expectations.

Another method of treatment by influencing the circulation of the parts and causing a redux hyperaemia is that by "interrupted circulation," which has been recently advocated by Dr. W. Ewart. The patient being recumbent, the limb to be treated, arm or leg, is raised and emptied of blood by pressure along the course of the veins. A protecting pad is placed around the thigh or upper arm, and the circulation is arrested by means of a piece of india-rubber tubing applied tightly in a single or double loop. The constriction is kept up for from thirty seconds to two minutes and is then suddenly relaxed, so that the limb becomes flushed with blood. The alternate constriction and relaxation are repeated three or four times at a sitting, at intervals of a few minutes, and the sittings may be repeated twice in the day. It is said that under the influence of this treatment the pain and swelling of the joints is usually greatly diminished, and that the relief obtained may be lasting. Moreover, in cases in which the joints are stiffened, passive movements can be carried out during the periods of arrested circulation, and so, even in crippling cases, improved mobility may be obtained.

The acute pains which sometimes result from the implication of nerve-roots when the vertebral joints are involved may be treated by blisters, and are sometimes conspicuously relieved by the application of the actual cautery near the spine.

In the early stage of sequels, when the trouble in the joints is no longer active, and when the constitutional symptoms have subsided, but before the deformities and contractures have become confirmed, massage, gentle passive movements, and exercises may prove of much service. Care must be taken to suspend such treatment for a time, when, as sometimes happens, there are signs of renewed activity in the damaged joints. At this stage, too, thermal treatment by baths and douches, aided by massage, is of special value. Hot-air and radiant-heat baths are also useful at this period. Later on, when the contracture-deformities are fully developed, they are irremediable. In the case of the knees, extension by weights, if well borne, will sometimes bring about straightening, but my experience of this treatment has not been encouraging, as the good effects are apt to be temporary, and usually do not compensate for the irksomeness of the treatment and the discomfort which it entails.

In exceptional cases surgical measures, such as excision of a knee-joint, may appear advisable, as in cases in which one knee is flexed as the result of a contracture, whereas the other knee has escaped without any serious damage.

Max Schüller strongly advocates incision of joints and removal of the hypertrophied synovial fringes in cases in which the joints are swollen and rendered more or less useless by their presence.

Two of the most important points in the treatment of rheumatoid arthritis have been left to the last. The first of these is the necessity of attention to any local trouble which may possibly have served as an infecting focus. The condition of the gums should be carefully investigated, pyorrhoea alveolaris, if present, should be suitably treated, and decayed stumps should be removed. Any local ulceration, such as ulcerated piles, should of course be treated, and in some few cases such local measures are followed by remarkable amelioration of the patient's general condition and by conspicuous improvement in the condition of the joints. Of no less importance is the efficient treatment of menorrhagia or other menstrual disorders. There can be no doubt that the presence of such disorders, by impairing the general health of the sufferers, tends materially to aggravate the articular disease, and that whilst they continue treatment more particularly directed to the latter has little chance of success.

To sum up, it cannot be too strongly insisted that rheumatoid arthritis is not a disease in which treatment is useless, but that by judicious treatment much may be accomplished; that the treatment followed should be adapted to the stage which the disease has reached in each individual case; that in the early stages the object aimed at should be the arrest of the disease, and in the later stages the minimising of its after-effects; that any plan of treatment embarked upon must, if it is to prove effectual, be persevered with irrespective of the absence of immediate good results. Patients who quickly tire of various plans of treatment in turn, are always trying new remedies and never persevere with

any, so that little hope can be entertained of a favourable result. The worst enemies of many such patients are well-meaning lay friends, who freely supplement and often counteract professional advice.

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Osteo-Arthritis

By A. E. Garrod, M.D., F.R.C.P.

The cases in which the joints are the seats of osteo-arthritic lesions, such as fibrillation and erosion of cartilages, lipping and eburnation of the articular ends of the bones, and osteophytic formation, although they resemble each other closely in their morbid anatomy, cannot be considered under a single clinical heading. Just as lumbago is a variety of muscular rheumatism, the characters of which are reproduced so faithfully in case after case that it calls for special description apart from other forms of myalgia, so also some of the local forms of osteo-arthritis, and especially the hip-joint affection of elderly people, may claim to rank as distinct varieties. It is indeed open to doubt whether the lesions of osteo-arthritis are due to any one specific morbid process, and are not rather a

series of changes which may occur in joints as results of various causes. In many instances they appear to originate spontaneously, or have the aspect of degenerative lesions associated with advancing age, or resulting from mere wear and tear. In other cases they date from, and seem to be attributable to, injury to the part. Lesions of this nature may occur in haemophilia after repeated attacks of intra-articular haemorrhage, and in tabes dorsalis and syringomyelia similar changes, but more extreme in degree, are attributed to impairment of the trophic functions of the nervous system. But between the several local varieties of osteo-arthritis, in which it is limited to a single joint or to a pair of corresponding joints, and the more generalised form of the disease, all intermediate stages present themselves. Thus, patients with *malum coxae senile* often have Heberden's nodes upon their fingers, or affection of the carpo-metacarpal joints of the thumbs.

Morbid Anatomy of Osteo-Arthritis.—The changes in the joints in osteo-arthritis are not confined to any one structure, but implicate alike the cartilages, bones, and synovial membranes.

At a very early period of the disease the articular cartilages assume a velvety appearance, due to the breaking-up of their ground substance into fibrillae, whilst the cells are lost. There is at the same time a heaping-up at the edges of the cartilages which is the initial stage of osteophytic formation. These changes were attributed by Cornil and Ranvier to a multiplication of the cells through the entire depth of the cartilages, and to the formation of capsules around them. They described how these capsules become distended and burst, discharging their contents into the articular cavity, and thus the ground substance becomes separated into filaments. At the edges, where the cartilage is overlapped by synovial membrane, the cells cannot escape into the joint cavity, and, accumulating there, give rise to chondrophytic and ultimately to osteophytic formation. The fibrillated cartilages become worn away, their central portions being the first to disappear, and the underlying bone is exposed. The bony surfaces thus exposed acquire a thin surface layer of great density, like ivory, and are described as eburnated, and later the polished surfaces become grooved by the movements of the bones upon each other. In the ivory-like surfaces there are minute perforations—exposed Haversian canals. The abrasion of the bones may reach an extreme degree in advanced cases, and by the wearing-down of the central portion of their articular faces on the one hand, and by the growth of peripheral osteophytes on the other, the mechanism of the affected joints may become profoundly altered, so that what was once a convex surface may become converted into a cavity, whereas the corresponding concavity may have become a convexity. The heads of the bones, as for example of the femora, may assume a flattened, mushroom form which can be clearly detected in skiagrams. Although the mobility of the affected joints may be almost abolished by the interlocking of osteophytes, bony ankylosis does not occur. There may be an extensive osteosclerosis of the deeper portions of the bones, which also may be made



manifest by the Röntgen rays, but in senile cases the cancellous tissue may be rarefied rather than condensed. The synovial membrane becomes thickened, and the synovial fringes tend to be hypertrophied. The tufts so formed may undergo fatty changes, or cartilaginous nodules may develop in them; moreover, such cartilaginous masses may have their pedicles torn, and may become loose bodies in the cavity of the joint. In the hip-joint the ligamentum teres becomes destroyed as the disease advances, and in joints with an intra-articular cartilage, such as the temporo-maxillary articulation, this also may be completely destroyed.

To sum up, the characteristic lesions of osteo-arthritis are, fibrillation and erosion of cartilages, the formation of bony lips and osteophytes, eburnation and abrasion of the articular surfaces of the bones, and the thickening of synovial membrane, with hypertrophy of the synovial fringes which are apt to become fatty or cartilaginous.

Heberden's Nodes.—Heberden's *Commentaries*, published in 1802, give a clear description of these "small tumours, about the size of a pea, which sometimes form near the third joints of the fingers," being, as he said, "rather disfiguring than inconvenient, although the movements of the fingers are somewhat hindered by them." That these enlargements of the terminal joints are due to bony outgrowths is readily made out by palpation, and this conclusion is confirmed by the Röntgen rays as well as by post-mortem evidence. Sometimes, in addition to the bony enlargement, small translucent cystic swellings are seen upon the postero-lateral aspects of the joints, at the summits of the nodes, and are probably herniae of the synovial membranes. When the change in the joints has reached an advanced stage, the terminal phalanges are deflected towards the radial side. The nodes are usually symmetrical upon the two hands, but as time goes on all the end-joints of the fingers are prone to be affected, and those of the thumbs may present a single nodose swelling on their radial sides. Although usually almost painless, Heberden's nodes may be seats of considerable pain, and there may be numbness and tingling of the fingers. The stiffness of the affected joints may also cause serious inconvenience by interfering with the finer movements of the fingers. In not a few instances such nodes are the only, or at least the earliest, manifestations of osteo-arthritis, but they often occur in association with other such lesions, or as part of a generalised form of the disease.

The question whether they stand in any direct relationship to gout has often been discussed, and different writers have expressed different opinions on this point. There can be little doubt that similar enlargements of the distal interphalangeal joints are sometimes seen in association with gouty troubles, and apart from any definite attack of uratic arthritis in the hands. These gouty manifestations, however, are apt to be limited to the end-joints of one or two fingers, are often asymmetrical, and are accompanied by an obvious swelling of the structures around the joints, which gives to them a more bulbous and less nodular appearance. It seems clear that in the great majority of instances Heberden's nodes

are osteo-arthritic lesions, which, to quote Heberden's words, "have certainly nothing in common with gout, for they are found in many patients who have no experience of that disease."

Osteo-Arthritis of the Carpo-Metacarpal Joint of the Thumb.—It is remarkable that this variety of osteo-arthritis has been so little noticed by writers upon this subject, for the carpo-metacarpal joint of the thumb is certainly among the seats of election of the morbid process. Not unfrequently this joint alone is attacked, and it is implicated in the majority of the more generalised cases. The lipping of the ends of the bones produces a conspicuous swelling over the joint, the osteophytic enlargement is clearly felt, and relaxation of the capsule and well-marked crepitus on movement are prominent features. The disease of this joint seriously impairs the usefulness of the thumbs, and it may be the seat of considerable pain. When one carpo-metacarpal joint is seriously affected the corresponding joint of the other thumb is usually implicated sooner or later.

Osteo-Arthritis of the Knees.—This, which is an extremely common affection of women about the menopause, usually comes under medical observation at an early stage owing to the importance of the articulations attacked. Some pain and stiffness are first felt on kneeling, or in going up or down stairs, and become aggravated as time elapses. At an early stage there may be no swelling of the affected joints, nor any palpable lipping of the bones. But when the patella is depressed with the thumbs whilst the joint is slowly flexed and extended a very distinct scrunch is felt, which differs in character from the synovial crackle so commonly felt in knees. The scrunch is well heard when the stethoscope is applied at the side of the patella, and it may here be mentioned that auscultation of joints as a clinical method is worthy of more general attention than it has received, and in many cases affords valuable evidence of the condition of the articular structures.

In later stages, if the disease progress, more obvious osteo-arthritic changes are observed, including lipping of the patella and the ends of the long bones, swelling and crepitus, and in many instances the presence of loose bodies in the articular cavity. These may become nipped in the movements of the joints, and sudden acute pain may result. The recognition of osteo-arthritis of the knee-joints in its early stages is particularly important, because when taken in time this affection proves by no means unamenable to treatment in many instances. It may occur as a strictly localised variety, although incipient Heberden's nodes sometimes bear additional testimony to the nature of the morbid process at work.

Osteo-Arthritis of the Temporo-Maxillary Joints.—Although much less frequently attacked by osteo-arthritis than by rheumatoid arthritis, the temporo-maxillary joints occasionally bear the brunt of the former disease. They may indeed be the only joints implicated in one of the more clearly defined localised varieties of the malady. When this is the case the movements of the jaw may be so seriously restricted that the

patient is obliged to take soft or liquid nourishment through gaps in the teeth. The inability to open the mouth is not wholly due to limitation of the range of movement of the temporo-maxillary joints, but depends in part on spasm of the masseters, as is shewn by the greater range of movement possible when the patient is under an anaesthetic.

Spondylitis Deformans.—The rigid condition of the spinal column thus designated is usually due to morbid processes which are to all appearance quite distinct from osteo-arthritis, and produce ossification of the spinal ligaments. The condition will therefore be discussed in a separate section (p. 39). Here it need only be said that the vertebral joints are not uncommonly the seats of osteo-arthritic lesions, that some stiffness of the cervical or lumbar spine is a common feature in the cases in which the disease is at all generalised, and that symptoms due to pressure on nerve-roots occasionally result.

The Hip-Joint Disease of Elderly People.—This is at once the most sharply defined and the most important of the localised varieties of osteo-arthritis. Its onset is often to be attributed to an injury, slight or severe, and it is peculiar in being considerably commoner in males than in females. In a large proportion of the cases the disease remains confined to one hip-joint, but a slighter degree of implication of the other hip is not uncommon, and occasionally both joints are equally affected. Not unfrequently Heberden's nodes or other osteo-arthritic lesions accompany the disease of the hip-joint.

Although usually a disease of late life, it is not rare between the ages of 50 and 60, and may be met with in patients who are even younger. Occasionally a hip-joint which has been the seat of disease in early life, but which has caused no active trouble for many years, once more becomes painful from osteo-arthritic changes.

Pain of various degrees of severity, and referred to the groin or even to the knee, and limitation of movement are the initial symptoms. In some instances, although there is much stiffness, pain is almost a negligible quantity. The affection which is most liable to be confused with osteo-arthritis of the hip is sciatica, and in some cases the diagnosis between them is no easy matter. Sometimes the two appear to coexist, just as there are often signs of affection of the shoulder-joint in connexion with brachial neuritis.

The chief diagnostic signs are the limitation of movements of the hip-joint and the distribution of the pain, which in osteo-arthritis does not extend below the knee and is often most severe in the groin and front of the thigh. Limitation of movement, when slight, may be masked by tilting of the pelvis, and care should be taken to avoid error from this cause. Useful evidence is often to be obtained by asking the patient to cross one leg over the other while in a sitting posture. When the hip-joint is implicated he may be unable to do this, or only able to do so by lifting the thigh with his hands. It must not be forgotten that the hip-joint lesions and sciatic pain sometimes co-exist. Arthritic muscular atrophy is usually well marked in malum coxae senile ;

the buttock may be flattened and the thigh conspicuously wasted. The knee-jerk is increased. In sciatica the wasting, when present, is more generalised and extends to the entire leg.

In advanced cases of the hip affection the movements of the joint are apt to be greatly restricted, and the restriction may be specially noticeable when abduction is attempted. Disease of both hips may make it impossible for the patient to bestride a horse. Even in comparatively early stages there may be apparent shortening of the limb, due to tilting of the pelvis; but in advanced cases there may be real shortening, from destruction of the head of the femur. In some cases the patient is conscious of a distinct scrunch or grating in the affected joint.

Baker's cysts are occasionally seen in association with osteo-arthritis of the hip, as with the same process in other joints. These collections of fluid, which may attain to great dimensions, stand to ordinary bursal swellings in the same relation as do false to true aneurysms. Morrant Baker, who first described these cysts, believed that the synovial fluid makes its way along the channel by which an ordinary bursa communicates with the joint, or forms a hernia of the synovial membrane. As the tension of fluid increases, escape from the sac into the surrounding tissues occurs, and in this way a collection is formed which is bounded by the muscles and other tissues amongst which it lies. Even when the cyst is far from a joint it is not safe to assume that it does not communicate with it.

The tendency of osteo-arthritis of the hip-joint is to progress uninfluenced by treatment, but when pain is severe in the earlier stages it usually becomes less intense as the disease advances.

Generalised Osteo-Arthritis.—No sharp line of division can be drawn between the local varieties of osteo-arthritis, even those which are apt to be limited to a single joint or pair of joints, and the generalised form, in which many joints present the characteristic lesions of this disease, for examples are met with of all intermediate degrees.

It is the generalised form which presents most superficial resemblance to rheumatoid arthritis, yet it is permissible to doubt whether, if the study of these maladies were approached *de novo*, apart from the trammels of tradition and inculcated notions, it would occur to any one to regard them as identical. Both are commoner in women than in men, both may implicate many joints large and small, both may attack the articulations of the spine and the temporo-maxillary joints, and both persist over long periods and cause disablement and deformities; but here the resemblance practically ceases, for the clinical and anatomical features of the articular lesions, and also their distribution, are very different.

The age-incidence of osteo-arthritis is later than that of rheumatoid arthritis, as is shewn by the following table, based upon a hundred cases taken without selection, save that all strictly localised cases were excluded. Had malum coxae senile been included, the proportion of males would have been considerably higher.

Age.

	Under 30.	30-40.	40-50.	50-60.	60-70.	Over 70.	Total.
Females . .	0	4	23	36	17	3	83
Males . . .	0	1	3	6	4	3	17
	0	5	26	42	21	6	100

From comparison of this table with that on p. 5 it will be seen that whereas the onset of rheumatoid arthritis is most frequent between the ages of 20 and 40, that of osteo-arthritis occurs most often between the ages of 40 and 60.

The clinical differences are very noticeable in the hands. The hand of generalised osteo-arthritis usually shews a combination of Heberden's nodes and enlargement and lipping of the carpo-metacarpal joints of the thumbs, and there is nothing comparable with the fusiform swelling of the proximal series of interphalangeal joints which is such a prominent feature of rheumatoid arthritis, in which disease the joints of the terminal row often escape. When the osteo-arthritic lesions attack the proximal interphalangeal joints the swelling is abrupt and nodular, and obviously due to osteophytic outgrowths, without much thickening of the capsules of the joints.

Muscular atrophy is a much less prominent feature of osteo-arthritis, and in it never reaches the extreme degree to which it often attains in rheumatoid cases. The deformities seen are not the results of muscular contracture, but are due to bony enlargement and such displacements as the radial deflexion of the terminal phalanges which is associated with Heberden's nodes.

Constitutional symptoms, the rapid pulse, and pigmentation of the skin are wanting in generalised osteo-arthritis; stiffness, pain, and disfigurement of the joints are the only symptoms complained of in most cases. No visceral lesions can be directly referred to osteo-arthritis, although in as far as the patients are often advanced in life they are apt to exhibit the degenerative changes which are common in later life. Any impairment of general health which is not attributable to intercurrent disorders is due to such causes as continuous pain and broken sleep rather than to any direct effects of the malady.

It cannot be supposed that the differences in the clinical and anatomical features of osteo-arthritis and rheumatoid arthritis are merely determined by the age of the patient. Characteristic examples of generalised osteo-arthritis are sometimes met with in comparatively young subjects, and in rare instances even in children. On the other hand, rheumatoid arthritis, with all its distinctive features, may commence in advanced life at a period when osteo-arthritic troubles are

much more common. Neither can the changes peculiar to osteo-arthritis be regarded as sequels of the rheumatoid lesions. The deformities which result from rheumatoid arthritis differ in character from the osteo-arthritic lesions, although in the end the cartilages may become completely destroyed and bony spicules may be thrown out from the articular ends of the bones. On the other hand, in cases of osteo-arthritis there is no preliminary acute or subacute attack; the lesions are destructive in character from the outset, and the differences in their distribution exclude the notion of direct sequence.

The **prognosis** varies greatly in different varieties of osteo-arthritis. In malum coxae senile the chance of material improvement is slight, and slowly progressive disability is the rule. In the knee-joint cases on the other hand, and in the more generalised forms, treatment, provided that it is initiated in the early stages and is carried out persistently, irrespective of immediate results, will often serve to arrest the progress of the disease, and although the damage already done to the articular structures cannot be removed, the pain and disability may markedly diminish. In not a few cases, however, no treatment has any obvious effect, and, speaking generally, favourable results are rather to be hoped for than promised. Even at the worst the outlook is less unfavourable than in severe cases of rheumatoid arthritis, for although many joints may be attacked, one after the other, the crippling produced is seldom so extreme as that which results from muscular contracture, and the incapacity induced is often more easily borne because the patients are usually more advanced in life.

The treatment of osteo-arthritis, at least in its early stages, is by no means of so little avail as is commonly supposed. In considering the results obtained attention must not be concentrated too closely upon the damaged joints, and even in cases in which there is little apparent improvement in the condition of these there may be evidence of arrest of a malady which was steadily advancing, or signs that its spread to fresh articulations is being limited.

The damage done by the disease is doubtless irreparable, but under the influence of suitable treatment joints which are only slightly affected may cease to be painful, and may apparently recover completely, although the physical signs which they exhibit may still be obvious. This is especially the case with the knee-joint affection, which is so common in women at about the period of the menopause. Such cases usually come under observation at a very early stage, when there is little or no swelling, but when the characteristic scrunch beneath the patella, which is probably due to erosion of cartilage, is clearly felt and heard. Such cases often do extremely well; no fresh joints may be attacked, and the knees cease to give trouble, although the scrunch persists. But it is never safe to promise much in osteo-arthritis, for some cases are wholly uninfluenced by treatment, and only a distinct improvement under its influence justifies a favourable prognosis in any given instance.

In some localised varieties, and above all in the hip-joint disease of elderly people, treatment is seldom of any real avail, even when the condition comes under observation in an early stage.

For the control of pain in osteo-arthritis aspirin often proves very efficient, but in many cases it fails to give any real relief. It is in this disease, and not in rheumatoid arthritis, that good effects are often obtained by the administration of iodide of iron and arsenic. It is, however, in the early stages that the effect of these drugs is best seen, and to obtain good results they must be persevered with for months, irrespective of obvious effects, but with suitable intervals and the omission of the arsenic from time to time. When such treatment is consistently carried out the disease often ceases to make any further headway, and there may be conspicuous improvement in the condition of the joints already attacked. It must not, of course, be expected that this or any other form of treatment will remove osteophytes or repair eroded cartilages.

The effects of thermal treatment are often conspicuously good in cases of osteo-arthritis, but it is by external application of mineral waters that such results are obtained, and it is in the early stages of the disease that the best results are got. The douche-massage treatment associated with Aix-les-Bains is especially serviceable to such patients, who may be advised to visit Aix, or the various British spas at which such treatment is carried out, such as Bath, Harrogate, and Buxton, to mention only a few of the more important resorts. Woodhall Spa also calls for mention as a watering-place at which osteo-arthritic patients are treated with success. Hot-air and radiant-heat baths often prove of service, and may give much relief from pain and be followed by increased mobility even when the disease has reached an advanced stage. In hip-joint cases spa treatment is of comparatively little avail, and the patients have often reached an age at which such treatment ceases to be desirable. In many instances the patient can only be recommended to make the best of his condition, and to adapt his surroundings as far as possible to his condition. Life on the flat is the goal to be aimed at.

Skilful massage is often useful in such cases, and by restoring the wasted muscles may increase the patient's powers of getting about. Forcible movement of the stiffened joints is not to be recommended in cases of osteo-arthritis. There are no fibrous adhesions to be broken down, and increased mobility is only obtained at the expense of further damage to the articular structures. By such treatment a moderately painful joint may be converted into an extremely painful one, and good results are quite the exception. Local applications, among which the linimentum potassii iodidi c. sapone of the British Pharmacopoeia is one of the most useful, often appear to give some relief, but in many cases, when the disease has advanced beyond a certain point, little can be done for the patients, who become in time more or less reconciled to their disabilities, and regulate their lives according to their remaining powers.

There is no reason to believe that dietary indiscretions have any

share in the causation of osteo-arthritis, nor that any special limitations of diet exercise any influence upon its course. If, as not infrequently happens in patients at the period of life when osteo-arthritis is commonest, there are gouty manifestations in addition, the diet should be regulated accordingly; but in cases in which there is no evidence of associated gout, a restricted diet often does harm, and the object aimed at should be to give the most nutritious diet that the patient's digestion allows.

A warm and equable climate adds materially to the comfort of the patients, and residence by the sea is apt to be followed by increased pain. The clothing should be suitable to the season and climate, and exposure to cold and damp should be avoided. In dealing with a chronic disease such as this, it is not necessary to impose rest, and the patients may be advised to use their damaged joints in moderation. They should, however, avoid such active use as entails pain lasting for hours after the exercise. Such limits they will, as a rule, impose upon themselves, unless, as is sometimes the case, the patients perseveringly provoke suffering under the impression that by so doing they are preserving the mobility of the diseased joints.

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A. E. G.

Spondylitis Deformans

By A. E. Garrod, M.D., F.R.C.P.

Synonym: *Rigidity of the Spinal Column.*

That pain and limitation of movement of the articulations of the spine are not unfrequent in the course of such articular diseases as gonorrhoeal rheumatism, rheumatoid arthritis, and osteo-arthritis is shewn in the description of those maladies. In these diseases, however, the spinal troubles are for the most part associated with widespread lesions of other joints, and, although they occasionally attain to unusual prominence, they are usually but minor features of the cases. It is entirely different with regard to the cases coming under the heading of

spondylitis deformans, in which rigidity of the spinal column is by far the most conspicuous feature, whereas the joints of the extremities may escape altogether or are only implicated to a very limited extent. It is only of quite recent years that these cases have received the attention they really deserve, but since 1896 they have formed the subject of a number of valuable monographs, in which their clinical features have been set out in a fairly complete manner. On the other hand, our knowledge of their pathology and morbid anatomy is still very imperfect; the few necropsies recorded have revealed what may probably be regarded as the final stages of the morbid processes at work, but have thrown comparatively little light upon the steps by which the observed lesions have been brought about.

It is still very doubtful whether the cases in question should be regarded as constituting a definite clinical and pathological condition, or whether they are examples of several distinct maladies. The recent impetus to their more complete study was given by the descriptions, by von Bechterew on the one hand and by Strümpell and Pierre Marie on the other hand, of certain minor groups of cases which they regarded as constituting distinct subgroups, and due to entirely different series of events. On the other hand, still more recent observations have tended to smooth away these differences; since cases of intermediate characters, which fill up more or less completely the interval between the two main groups, have been described, and the general opinion at the present day inclines to the view that the conditions described by von Bechterew and by Strümpell and Pierre Marie respectively are rather varieties than distinct morbid species.

Etiology.—Rigidity of the spine occurs in the great majority of instances in males, and sometimes appears in quite early life. Indeed some observers have considered that it is most frequently met with in young men.

Its causation has been assigned to a number of factors, such as gonorrhoea and syphilis, hereditary tendencies, and injury. In some of von Bechterew's cases the onset of the disease followed somewhat closely upon an injury to the back, but in connexion with such a cause, it must not be forgotten that gross damage to the spine may result in a condition which simulates somewhat closely that under discussion.

The cases of senile kyphosis with spinal rigidity, not unfrequently seen in country lanes and village streets, in aged agricultural labourers, seem to form a special class.

Morbid Anatomy.—The few published post-mortem records shew that the main anatomical features of the condition are bony ankylosis of the intervertebral articulations and the ossification of the spinal ligaments. This ossification is not limited to the ligamenta flava and other ligaments in contact with the spinal column, which may enclose it as in a rigid tube of bone, but also invades other ligaments, such as the interspinous, which may unite the several spines by bridges or flying-buttresses of bone. The capsules of the intervertebral joints, of the joints between

the ribs and vertebrae, and even of the hip-joints, may be in like manner ossified, in addition to the bony ankylosis of the articular surfaces. The intervertebral discs may be intact even when enclosed in a bony sheath, or may be absorbed to a greater or less extent; occasionally there is true bony ankylosis of the bodies of the vertebrae. In spite of so much formation of new bony material, osteophytes such as are met with in osteo-arthritic joints are usually absent, and as has already been mentioned the foramina through which the spinal nerves emerge are but little encroached upon.

In the cases described by von Bechterew the upper dorsal and cervical spine was rigid and conspicuously bowed. There was marked flattening of the thorax and fixation of the ribs. There was no compensatory lordosis of the lower portions of the spine, and the patients stood leaning forward and with knees slightly bent. Nerve-root symptoms of varying degrees were present, such as causalgic pains, paraesthesia, impairment of sensation in places, and slight degrees of atrophy of the muscles of the arms, and of the scapular muscles.

In one of his cases, examined after death, the changes in the affected portion of the spine were unequally distributed. Some of the intervertebral discs were wholly destroyed and others atrophied. There was bony ankylosis of the anterior parts of the bodies of many of the vertebrae, whereas in the lower dorsal and lumbar regions the mobility of the vertebrae upon each other was not impaired. The nerve-roots of the corresponding portion of the cord shewed evidences of degeneration, which were far more pronounced in the posterior roots and extended to the cord itself and especially to the posterior columns. In these regions the pia mater over the posterior aspect of the cord shewed inflammatory thickening. There was no narrowing of the foramina which could have produced pressure on the nerve-roots. Von Bechterew is inclined to regard the nerve lesions as primary, or rather as secondary to a local inflammation of the spinal meninges, and the spinal rigidity as a secondary effect. The kyphosis he attributes to paresis and wasting of muscles, and the absorption of intervertebral discs and ankylosis of the vertebrae he regards as resulting from the kyphosis.

To the second main group of cases Pierre Marie has given the name of Spondylose rhizomélique because the articulations of the roots of the limbs, the hip- and shoulder-joints, are usually implicated to a greater or less degree. The lower portion of the spine is earliest affected in these cases, but a similar flattening of the chest and fixation of the ribs is often present. At a post-mortem examination described by Léri the changes described in the general sketch of the morbid anatomy of the condition were met with — including ossification of ligaments and ankylosis of articulations. The ribs were completely ankylosed to the vertebrae. There were no appearances of disease in the spinal cord or its membranes. A very complete account of the spine and hip-joints of such a case has been published in this country by Hilton Fagge.

A. Magnus-Levy, who holds that these two forms cannot be sharply

differentiated from each other, describes cases which in most respects conform to von Bechterew's class, but in which the kyphosis was slight or absent, and points out that nerve-root symptoms are met with in both forms but are not constant in either. Transitional cases have been described by W. Anschutz and others.

Clinical Aspect.—Taking the cases as a whole the most conspicuous clinical features are the following:—The spinal column is rigid either throughout its entire length, or in part, and in the latter case the part affected may be the upper or lower portion only. In extreme cases all mobility is lost, the atlanto-axial joints may become fixed, mobility of the spine in every direction, antero-posterior and lateral flexion as well as rotation, is abolished, and the spine as a whole may be rigidly attached to the sacrum. There is often a considerable degree of kyphosis of the upper portion of the spine without compensatory lordosis, but such bending is by no means always present, and for not a few cases the name of "poker-back" is not unsuitable.

Of the joints of the limbs, the hips and shoulders are most often affected. Their movements become greatly limited, the thighs tend to be somewhat flexed upon the pelvis and are slightly adducted, and in extreme cases the hips become completely ankylosed. The knees too may become affected, the shoulders less often and to a less degree than the hips, and there is often limitation of movement of the temporo-maxillary joints. The changes in the joints of the extremities rather resemble those described by Ziegler under the name of arthritis chronica ankylopoietica than the familiar changes of osteo-arthritis.

Various symptoms referable to the nervous system are apt to be present, and these are such as might well result from pressure upon the roots of the emerging nerves. Autopsies, however, have not shewn the narrowing of the intervertebral foramina that might be expected, so that the manner in which the pressure on the nerve-roots is brought about is still in need of further elucidation. From my own experience I should assign to these nerve-root symptoms a more prominent place in the symptomatology of the disease than is usually given them in the published descriptions. Among such symptoms pain radiating along the course of the nerves concerned is the most prominent and most distressing, and some patients who apply for relief from such pains are unconscious of the rigidity of their spines. Paraesthesiae of various kinds and a greater or less degree of muscular atrophy also call for mention.

A conspicuous feature in the majority of the cases is flattening of the thorax in the antero-posterior direction and immobility of the ribs. As a result of this the breathing becomes almost wholly abdominal. It has been definitely proved that, in some cases at any rate, this immobility of the thorax is due to ankylosis of the joints of the ribs with the spine, which forms part of the morbid process in the latter.

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THE ARTICULAR LESIONS OF INFECTIVE DISEASES

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It is now generally recognised that inflammation of joints occurs as a phenomenon of a large number of infective maladies, and that no sharp division can be drawn between a disease such as rheumatic fever, in which, in adult life at least, arthritis is the dominant manifestation, and others, such as measles and mumps, in which its occurrence is rather a clinical curiosity; between these extremes every grade is met with. This article deals with certain infective arthritides which are sufficiently common to call for individual description.

As the result of the manner in which our knowledge of articular lesions has gradually been extended, these varieties have, one by one, been differentiated off from the rheumatic category, but to not a few of them the name of rheumatism still clings, or they are spoken of as pseudo-rheumatic affections. Their recognition has been largely due to the labours of the French school, for some members of which this subject has long had special attractions. In more recent years bacteriological investigations have conferred a much higher degree of accuracy upon such researches, and have made accurate differentiation possible on other than clinical grounds.

It cannot be assumed that an arthritis occurring in the course of a zymotic malady is of necessity due to the specific micro-organism of the disease in question, either directly by its invasion of the articular structures or indirectly through its toxins. The lesions may be due to secondary infections, and even in cases in which the specific organism is found in the joints, other secondary bacteria may be present in preponderating numbers. Of the varieties of arthritis to be considered, the so-called gonorrhoeal rheumatism may justly take the first place, not only on account of its frequency, but also because when the gonococcus has invaded the organism the resulting articular lesions dominate the

clinical picture as often and to as great an extent as do those of rheumatic fever.

Gonorrhoeal Rheumatism.—Whereas in the great majority of instances a gonorrhoeal infection remains limited to its primary seat or to neighbouring structures, to which it spreads by direct extension or by the lymphatic channels; in a small proportion of cases a systemic infection with the gonococcus results, the organism passing into the blood-stream and being conveyed to distant parts of the body. A number of lesions, articular and visceral, may then be produced, and to the group of symptoms presented in such cases it is convenient to apply the time-honoured name of gonorrhoeal rheumatism. It must be premised, however, that this use of the term gonorrhoeal rheumatism gives to it a much wider significance than it formerly had, but it is difficult to draw any hard-and-fast line between cases in which there is but slight constitutional disturbance, the local phenomena being confined to such structures as the joints, tendon-sheaths, fasciae, and the most acute cases of gonococcal septicaemia and pyaemia, seeing that examples of every intermediate degree of severity occur. The case is strictly parallel to that of true rheumatism, which, originally regarded as an articular disease, is now recognised as a systemic infective malady, arthritis being one only of the complications.

That arthritis occurs in association with gonorrhoea was recognised by Selle and by Swediaur as long ago as the end of the eighteenth century, but for a long time the nature of the association remained doubtful. It was suggested that the disease was true rheumatism modified in its characters by the presence of gonorrhoea, and even that the arthritis had a reflex nervous origin, and was urethral rather than gonorrhoeal. Only comparatively recently has its true nature been established, but it may now be asserted that of diseases which specially implicate the joints it is that of which our knowledge is the most satisfactory and most nearly complete.

Etiology.—The necessary precursor of an attack of gonorrhoeal rheumatism is a gonorrhoea, but it is in no way essential that the original infection should be genital. The articular and other manifestations may equally follow an ocular infection. They have been repeatedly observed in young infants suffering from purulent conjunctivitis, and have followed the inoculation of the conjunctivae for the treatment of granular lids. Poncet described a remarkable case in which the inoculation of the conjunctiva of each eye was followed on each occasion by an attack of gonorrhoeal rheumatism. The contrast between the extreme frequency of gonorrhoea and the rarity of gonorrhoeal rheumatism admits of more than one possible explanation. The special liability to a return of the disease shewn by a person who has once had gonorrhoeal rheumatism if again infected is well established, and suggests that the liability is inherent in the individual. That this liability is not innate is, on the other hand, suggested by the equally well-established observation, that the systemic infection may first occur during a second or third attack,

and may then recur with each subsequent attack; but this does not exclude the possibility that in such a patient a pathway for the blood infection is opened which renders the subsequent entry of the gonococcus into the circulation comparatively easy. Buschke suggests that the occurrence of gonorrhoeal rheumatism is to a large extent dependent upon a special virulence of the organism in the cases in which it occurs, and in support of this quotes instances in which individuals infected one from the other have both shewn the articular lesions. The length of the interval between the original infection and the appearance of the articular lesions varies widely in different cases. In some the interval is as short as a week, or even less; three or four weeks commonly elapse, but the period may even be extended to months, and at the time when the joints and other structures are attacked a slight gleet may be the only remaining evidence of the local lesion. In Poncet's case, referred to above, the arthritis followed a month after the inoculation of the first, and seven days after that of the second eye. It is obvious that when once the local affection is completely cured, and the gonococcus has died out, there can be no relapse, apart from a fresh infection; but in some cases, although the gonorrhoea is apparently cured, there remains a smouldering infection in the prostatic portion of the urethra, so that occasionally relapses occur apart from reinfection, and after long periods of immunity, in patients who had believed themselves to be entirely cured of the urethral lesion. The diagnosis may thus be very difficult, or even remain doubtful, in cases with articular and other lesions strongly suggesting a gonorrhoeal origin, but no evidence of urethral disease. The influence of external conditions in determining the seats of the local manifestations of systemic diseases is apparently potent, and it is not improbable, as some authors have maintained, that such a cause as an injury may determine an articular attack, or that, as in other articular diseases, a joint formerly damaged may be the first to become the seat of the arthritis. In most cases, however, no satisfactory evidence of the action of such concurrent causes is forthcoming.

Incidence.—The great majority of cases of gonorrhoeal rheumatism occur in men, and even when allowance is made for the greater frequency of gonorrhoea in men, their liability to systemic infection would appear to be much greater. The female sex is, however, by no means immune, and it is probable that the disparity is not quite so great as appears at first sight. No conclusions can be drawn from the age-incidence of gonorrhoeal rheumatism, since it is determined solely by that at which gonorrhoea is usually acquired. Although the great majority of cases occur in young adults, it is sometimes met with in later life, and the group of cases following ophthalmia neonatorum proves that even early infancy is in no way exempt.

Pathology.—It is unnecessary in the present state of our knowledge to refer further to the earlier views as to the nature of gonorrhoeal rheumatism. We have seen that the symptom-complex here presented is characteristic and unlike that of any other disease, a point to which

Fournier first clearly directed attention; the malady has the character of a definite specific disorder, comparable to true rheumatism, but essentially different from it. The generalisation of an infective disease, which in most instances remains localised, may be due either to the dissemination of the specific organism, or to systemic poisoning by the chemical products of its growth. The available evidence points to the former as the correct interpretation in this instance. It is easier to explain upon this view why of the many sufferers from gonorrhoea, a few only present gonorrhoeal rheumatism; and, since Petrone and Kammerer first investigated the subject, many observers have sought for the gonococcus in the synovial fluid from the affected joints and in the vegetations situated upon the endocardium in cases fatal from infective endocarditis. The evidence which they adduce is of varying degrees of cogency, but one by one the special properties of the gonococcus have been recognised in the organisms obtained from these situations.

It must now be regarded as established that, in some cases at any rate, the fluid obtained from the joints both of infants and adults, the blood, the pleural exudation, and the cardiac vegetations contain diplococci which present the characteristic shape of the gonococcus; they lie for the most part within the leucocytes, are decolorised by Gram's method, and grow readily upon a mixture of human blood-serum and agar, whereas they refuse to grow upon any ordinary culture medium. Indeed, the accounts given by von Leyden, Bordoni-Uffreduzzi, and Thayer and Blumer definitely prove the dependence of the above-mentioned lesions upon the dissemination of the gonococcus.

Even in the inflamed joints, however, the gonococcus has not been found so constantly as might be expected, and there is reason to believe that it is present in the synovial fluid in the early stage only of the arthritis. Moreover, the synovial fluid is probably not the best material in which to seek for the organism, which would, perhaps, be found in greater abundance in the soft tissues of the inflamed joints. In cases of gonococcal septicaemia, with endocarditis, the gonococcus has been repeatedly found in blood removed during life by Thayer and Blumer, Ahman and others, and recently by Dr. Horder.

General Symptoms.—The amount of constitutional disturbance accompanying gonorrhoeal rheumatism is very variable. In cases of gonorrhoea without generalised lesions there may be febrile disturbance and other symptoms, which have been ascribed to absorption of toxins from the primary focus; and, on the other hand, in cases in which the joints are affected the elevation of temperature may be slight. When the articular lesions are acute rheumatic fever may be simulated; and thus there is every grade of fever and illness met with, culminating in the profoundly grave symptoms met with in fatal cases of gonorrhoeal septicaemia and pyaemia.

The Arthritic Lesions.—Gonorrhoeal arthritis is more apt to occur in the larger than in the smaller joints. It may be confined to a single articulation, or may implicate almost every joint. Three or four joints

are commonly attacked in the course of the disease. The following table, compiled from those of Foucart, Brandes, Rollet, and Fournier, shews the relative frequency of the several joints affected in a total of 119 cases :—

Knee	attacked in	83	cases
Ankle	„	32	„
Fingers and toes	„	23	„
Hip	„	16	„
Wrist	„	14	„
Shoulder	„	12	„
Elbow	„	11	„
Temporo-maxillary joint	attacked in	6	cases
Metatarsus and tarsus	attacked in	5	cases
Sacro-iliac joint	attacked in	4	cases
Sterno-clavicular	„	3	„
Chondro-costal joints	„	2	„
Tibio-fibular joint	„	1	case

The table illustrates several points of interest. The quite exceptional liability of the knee is a matter of every-day experience, but it is surprising to find that this joint is attacked almost as frequently as all the other joints taken together. Further, certain articulations which are little liable to most other forms of arthritis, such as the sacro-iliac and chondro-costal synchondroses, the sterno-clavicular and tibio-fibular joints, are subject to gonorrhoeal arthritis. The liability of the temporo-maxillary joint also is a diagnostic point from rheumatic fever, but not from rheumatoid arthritis and osteo-arthritis.

The clinical features of the articular lesions differ widely in different cases. Sometimes there are pain and stiffness of joints with no obvious swelling. In some cases, in which the arthritis is of an asthenic kind, there is synovial effusion with little or no periarticular swelling. In more acute cases there is more periarticular swelling, local heat, and even a pink blush over the joints. Lastly, one or more joints may be the seat of a very acute arthritis with redness, much heat, and synovial distension so great as to call for paracentesis. Suppuration may even occur in such a joint. This, which is certainly rare in gonorrhoeal rheumatism, may sometimes be attributable to a mixed infection, but the synovial fluid is often semipurulent, and seeing that the gonococcus is a pyogenetic organism, it is perhaps surprising that suppuration does not more often occur. Kienböck has pointed out that the Röntgen rays reveal a rarefaction of the bones in the neighbourhood of the inflamed joints, which renders them unduly transparent, as in rheumatoid arthritis (see p. 11).

The arthritis has not the shifting character of that of rheumatic fever, and is much more persistent. Its duration is measured by weeks rather than by days, and fresh joints are liable to be attacked, whilst in others the inflammation is waning. Teno-synovitis with effusion into tendon-sheaths is common, and bursae, such as those of the patella and olecranon, are liable to become inflamed, distended, and swollen. The pain varies with the severity of the arthritis, and with the degree of synovial distension. It is apparently less severe than that of gout or rheumatism, save in the exceptional cases referred to above ;

and patients who have suffered both from gonorrhoeal and from true rheumatism describe the pain as differing in character in these two diseases. Muscular atrophy occurs, as in other forms of articular disease, but is only exceptionally a conspicuous feature.

Although it often persists so long, and is but little influenced by treatment, the tendency of gonorrhoeal arthritis is towards recovery. In the great majority of instances an attack of this disease does not result in any permanent damage to the structures affected, or only leaves behind it fibrous adhesions, such as can be broken down by massage and passive movements, or by forced movements under an anaesthetic. The commonest sequel is flat-foot due to the yielding of the ligaments and plantar fasciae in cases in which the foot and ankle have been attacked. Yet the prognosis is by no means wholly favourable. Some patients are left in a truly pitiable condition, with many joints firmly ankylosed, and muscles greatly atrophied, their state recalling that which very rarely follows rheumatic fever, the *rhumatisme fibreux* of Jaccoud. In such cases there has usually been a series of relapses, due to an uncured infection of the deep parts of the urethra. Moreover, there is good reason to believe that gonorrhoeal rheumatism, which is certainly apt to attack the vertebral as well as other joints, plays a part in the causation of the rigid condition of the spinal column, known as spondylitis deformans, an affection the pathology and etiology of which are still very obscure (see p. 44).

Occasionally joints, and especially knees, which have been the seats of gonorrhoeal arthritis exhibit the remarkable phenomenon known as intermittent hydrarthrosis (see p. 60), and become distended with fluid at fixed periods, which recur with almost mathematical precision. Lastly, there may develop a condition of many joints, clinically indistinguishable from rheumatoid arthritis, which results in the crippling and deformities due to muscular contracture, so familiar as sequels of that disease.

Pain in the heel calls for special mention, as an accompaniment or sequel of gonorrhoeal rheumatism. It is a pain situated beneath the os calcis, and brought out by the pressure of the foot upon the ground. In some cases the seat of pain appears to be the insertion of the plantar fascia, but sometimes it is due to a gonococcal inflammation of the periosteum of the os calcis. In any case in which such pain is complained of a gonorrhoeal origin must be excluded, but it is not uncommonly due to gout, and in some cases neither of these causes can be shewn to have a share in its production.

Affections of Fasciae and Muscles.—Pains provoked by movement, and indistinguishable in their characters from the so-called muscular rheumatism, are common, but among the rarer manifestations of gonorrhoeal rheumatism is an acute non-suppurative myositis, which is of special interest. Usually, but by no means invariably, the affected muscle is in the neighbourhood of, but not in actual contact with, an inflamed joint. Thus, associated with an arthritis of the knee there may be myositis of the gastrocnemius, or of the biceps or triceps in association

with affection of the shoulder-joint. The inflamed muscle is swollen, acutely tender, and the skin over it may be hot and reddened, but Lorenz describes the tenderness as usually less than in other forms of acute myositis. The muscle may feel as hard as wood, and the joint which it controls is held in flexion or extension, according to the action of the affected muscle. The stage of acute inflammation does not last long, but the muscle is left weak and somewhat wasted for some time after the myositis subsides. Cases of this kind have been described by Sir F. Treves, Dr. Sidney Phillips, Leube, Lorenz, and others; and I have seen a case in which the myositis occurred in the neighbourhood of an inflamed joint, and also at a later stage apart from any articular lesion.

J. E., aged twenty, had acquired gonorrhoea three months before he was first seen. Five weeks from its onset he had pain in both knees and in the left shoulder-joint. When he presented himself in the Casualty Department of St. Bartholomew's Hospital there were signs of effusion in the left shoulder-joint, the left arm was held in the flexed position, and the biceps humeri was swollen and as hard as wood throughout its entire length. The muscle was exquisitely tender, and there was considerable local heat; its inflamed condition precluded any movement at the elbow-joint, which was bent at a right angle, but was not itself involved. The patient was treated as an out-patient, and the myositis subsided. Three weeks after his first attendance he was admitted with a similar affection of the triceps of the other arm, which, unlike the previous myositis, could not be attributed to local extension, as the joints of the right arm were quite unaffected. This also subsided under treatment, but both the muscles attacked remained somewhat atrophied.

Manifestations in the Nervous System.—Sciatica either of the neuralgic form or a true sciatic neuritis is a well-recognised accident of gonorrhoeal rheumatism, but, as Eulenberg has pointed out, the affection is not usually strictly limited to one or both sciatic nerves. Brachial neuritis also occurs, but is much less common. The neuritis is usually in the neighbourhood of an inflamed joint, but, like myositis, may appear in a limb in which there is no arthritis. The muscles of the affected area may waste, and other trophic phenomena may ensue. Sometimes there may be conspicuous tenderness and palpable swelling of the inflamed nerve-trunks. In a remarkable case recorded by Lazarus, a temporary paralysis of the abductors of the vocal cords came on in the course of gonorrhoeal rheumatism. Lazarus considered that this was probably due to neuritis of the recurrent laryngeal nerves, and that the fixation was not due to a crico-arytaenoid arthritis, nor to perichondritis of the arytaenoid cartilages. Kienböck, who has collected many recorded examples of nervous lesions in gonorrhoeal rheumatism, and has himself observed others, believes that neuritis is a potent cause of muscular atrophy in this disease; this atrophy is not uncommon in areas so far removed from inflamed joints that it cannot be referred to the ordinary arthritic form. In some cases the intense nerve pains are referable to the implication of the vertebral joints and the secondary affection of the nerve-roots.

Kienböck also refers to cases of generalised neuritis without any articular lesions, and resembling that which results from toxic influences. Although he considers that in some of the recorded cases of this kind a gonorrhoeal origin is highly probable, he does not regard them as so obviously of this nature as the more local neuritic lesions referred to above. Not only the peripheral nerves but the spinal cord may be implicated, and gonorrhoeal paraplegia of spinal origin may be present, In such cases, which have been recorded by von Leyden, Sir W. Gowers, and others, the lesion present is usually a meningo-myelitis, and von Leyden believes that a large proportion of the cases upon which the conception of reflex urinary paralysis has been built up were probably, whilst some were certainly, of this nature. He, however, definitely excludes the cases in which the paralysis is associated with calculous lesions.

Ocular Lesions.—The affections of the eye in gonorrhoeal rheumatism are comparatively common, and take the form of conjunctivitis or scleritis, and iritis. The conjunctivitis, which is much more frequent than iritis, must be carefully distinguished from the purulent ophthalmia due to direct gonococcal infection of the conjunctiva. It is present at some stage in a considerable proportion of the cases, and appears, like the joint lesions, to be one of the manifestations of the systemic infection. Iritis, when it occurs, usually attacks the two eyes in succession or simultaneously. It is of the serous or "rheumatic" variety.

The following case offers a well-marked example of gonorrhoeal rheumatism with ocular lesions: a man of twenty-three years of age acquired gonorrhoea in June 1887. Three weeks later he had pain in the feet, followed by swelling of the left foot and ankle. Later both knees became swollen. The discharge ceased in October, according to the patient's statement. In January 1888, at which time he first came under observation, the joints were still painful, and there was some degree of flat-foot. He then had well-marked conjunctivitis, as well as iritis of the right eye. In February, after the right eye had entirely recovered, left iritis set in.

Fournier met with ocular lesions in fifteen out of thirty-nine cases, but the joints were not usually affected contemporaneously with the eyes. He quotes a case in which four attacks of gonorrhoea within five years were followed, in the first instance, by ocular lesions only; in the second and third instances by affections of the eyes and joints, and in the fourth by arthritis alone.

Cardiac Lesions.—Although pericarditis, endocarditis, and myocarditis have no place in the clinical picture of gonorrhoeal rheumatism in any respect comparable with that which they occupy in connexion with rheumatic fever, all these lesions do occasionally present themselves. An extreme case of gonorrhoeal myocarditis has been described by Councilman, and it has been clearly demonstrated by a number of observers, including Thayer and Blumer, Ahman, and Horder, that the gonococcus is one of the organisms which can and do produce malignant endocarditis. In addition to cases of fatal gonococcal septicaemia, in which

the presence of endocardial lesions swarming with gonococci can be demonstrated after death, there are others in which a cardiac murmur, apparently organic, appears during an attack of gonorrhoeal rheumatism, from which the patient makes a good recovery. Such cases suggest that gonococcal endocarditis is not invariably fatal, but direct proof of this is, naturally, not forthcoming.

On the other hand, infective endocarditis in the course of gonorrhoeal rheumatism is not invariably due to the gonococcus, but may, as in a case recorded by Ely, be due to a secondary infection with another organism.

Pleurisy must undoubtedly be regarded as one of the lesions of gonorrhoeal rheumatism. It may be "dry" or accompanied by effusion. A particularly convincing case of pleurisy with effusion, in which the gonococcus was cultivated from the pleural fluid, has been recorded by Bordoni-Uffreduzzi. The cultivated organism produced urethral gonorrhoea in a healthy man who volunteered to be the subject of the experiment.

Diagnosis.—The presence of active gonorrhoea or of gleet usually renders the diagnosis of gonorrhoeal rheumatism easy; indeed, the clinical features of the joint lesions themselves often suggest the correct conclusion, which may be confirmed by the presence of a urethral discharge. On the other hand, it is not safe to conclude that arthritis in a patient with gonorrhoea is necessarily gonorrhoeal. It is probable that the disease, at any rate in the earlier stages, is not infrequently mistaken for true rheumatism, especially when it occurs in women; but suspicion is soon aroused by the absence of marked fever, or by the obstinacy of the arthritis, which shews no disposition to yield to the salicylic treatment. Conjunctivitis, iritis, or arthritis in the temporo-maxillary or sterno-clavicular joints, in the course of a supposed attack of rheumatic fever, should always suggest the possibility of a gonorrhoeal origin; but it must not be forgotten that rheumatoid arthritis, in which the temporo-maxillary joints are more liable to suffer, in its more acute forms may also be mistaken for rheumatic fever.

Prognosis.—The prognosis on the whole is favourable; a fatal termination is extremely rare; and recovery, although long delayed, is as a rule complete. Nevertheless in giving a prognosis the risk of fibrous ankylosis of the affected joints, the occasional occurrence of gonococcal septicaemia, and also the possibility of the subsequent development of rheumatoid changes, should always be remembered. The patient should be emphatically warned that a fresh gonorrhoea is almost certain to be followed by a return of the articular disease, and that each successive attack is apt to be more severe, even more refractory to treatment, and more likely to cause permanent disablement.

Treatment.—As Sir James Paget taught, long ago, the essential point in the successful treatment of gonorrhoeal rheumatism is the cure of the primary lesion, and this, when of long standing and in the posterior part of the urethra, may be no easy task. So long as a focus remains from which gonococci may be again disseminated, treatment directed



against the pseudo-rheumatic lesions has little chance of proving lastingly successful. No known drugs have any conspicuous effect upon the articular lesions. Sodium salicylate is of little or no avail, and though aspirin often relieves pain, it is upon iodides only that reliance can be placed.

Rest is important in this as in other forms of acute or subacute arthritis, and should be enforced even when pain does not render it obligatory. With rest and time the inflamed joints usually recover completely, but it must be recognised that in this disease there is a considerable tendency for fibrous adhesions to be formed. In severe cases one or more joints may be intensely swollen and tightly distended with fluid, and, as has been already mentioned, actual suppuration may occur. Puncture is occasionally required for the relief of extreme tension, and some surgeons have strongly advocated arthrotomy and drainage at an early stage of the arthritis. Thus, Thiery (1892) recorded cases successfully treated in this manner, and O'Conor (1897 and 1899) advocated the general employment of these measures in gonorrhoeal rheumatism. There can be no question of the value of this plan of treatment in suitable cases, but these authors appear to take an unduly gloomy view of the prospects of ordinary cases, and to over-estimate the risks of fibrous ankylosis and of destructive changes in the joints. On the other hand, in not a few cases, the ultimate results would probably have been far more satisfactory if surgical measures had been resorted to at an early stage.

The method introduced by Bier, and extensively employed in Germany at the present day in the treatment of various morbid conditions, has yielded good results in this disease. The method has already been described in connexion with rheumatoid arthritis (p. 28). The hyperaemia induced by an india-rubber bandage lightly applied above the affected joints has been maintained for various lengths of time daily, varying from ten or twelve to twenty-two hours in the twenty-four, to two applications daily at first for a few minutes only, and later for longer periods up to three hours. This method is said to bring about a rapid relief of pain, and absorption of the effusion in the joints and in the neighbouring structures. Hirsch regards the treatment as valuable from its simplicity and the ease with which it is applied, but unlike some others who have tried it, did not find that it shortened the duration of the illness; nor would such local treatment be expected to exert any profound effect upon a systemic infection, although the absorption of the effusion may well preserve the joints from the more permanent damage that they might otherwise sustain.

The attempt to combat gonorrhoeal rheumatism with an anti-gonococcic serum is clearly indicated, and has already given encouraging results. Thus, Torry prepared an anti-gonococcic serum with which J. Rogers obtained very satisfactory results in the large majority of the cases in which it was injected hypodermically, some of the cases so benefited being of the more chronic and prolonged varieties. Un-

fortunately the serum has no marked effect upon the primary local trouble; Torry is inclined to ascribe this to the quantity of immune bodies which is able to cope with the comparatively few gonococci of chronic infections, being insufficient to destroy the large numbers present in the urethra in acute gonorrhoea. Drs. Soltau Fenwick and Parkinson have obtained excellent results with rectal injections of a polyvalent antistreptococcic serum in gonorrhoeal rheumatism and even in gonococcal septicaemia with endocardial murmurs. In their two cases of septicaemia and endocarditis recovery was complete so far as the joints were concerned, although the state of the patients appeared at one period to be desperate. Injections of 10 c.c. of the serum were administered daily per rectum for various periods in different cases. In a case of simple gonorrhoeal urethritis a very satisfactory result was obtained without any other treatment. Other serums, such as the antidiphtheritic, a specially prepared anti-gonococcic serum, and normal horse-serum in 20 c.c. doses, gave no relief in such cases. In each case the presence of a gonorrhoeal infection was established by bacteriological methods.

The slighter degrees of stiffness which not infrequently persist after the inflammatory changes in the joints have subsided may usually be removed by massage and passive movements, and hot-air or radiant-heat baths and the other methods of treatment carried out at health resorts prove of great service in removing such sequels of gonorrhoeal rheumatism. When firmer fibrous adhesions have formed more energetic measures, such as movement under an anaesthetic, may be necessary. In any case in which the condition of the joint is such that complete ankylosis is threatened, arthrotomy and drainage will probably be resorted to at the present day; but if for any reason this operation be decided against, care must be taken that if ankylosis should ensue the position of the limb may be the most favourable that is possible.

THE ARTHRITIS OF DYSENTERY.—That affection of joints is by no means rare in dysentery has long been known, and Dewevre has called attention to a reference by Sydenham to the occurrence of articular and muscular pains, like those of rheumatism, in cases observed during an epidemic in 1672. In the literature of the eighteenth century a number of references to these complications are found.

That arthritis is met with in bacillary dysentery is undoubted, but I have been unable to find any definite statement as to its occurrence in the amoebic form. In different epidemics the proportion of cases in which lesions of the joints occur varies considerably, but a common proportion appears to be 3 to 4 per cent.

Bacteriology.—I have not found any record of the detection of Shiga's bacillus in the inflamed joints. Remlinger (1898) failed to find any organisms in the articular fluid, and Salle was not more successful in 1900, but in 1903 he found the colon bacillus in the synovial fluid from the knees in two cases, and the same organism in the inflamed con-

junctiva of another patient. Major Beveridge, who described dysenteric arthritis among our soldiers in the South African War, 1899-1902, isolated and cultivated a small Gram-negative diplococcus from the peripheral blood of the patients when the articular symptoms were at their height, whereas in control cases of dysentery, in which the arthritis was absent, the peripheral blood proved sterile. He does not appear to have made any bacteriological examination of the synovial fluid.

Symptoms.—Arthritis cannot be regarded as a grave accident of the disease, seeing that it is most commonly met with in mild cases, and is of short duration, leaving no permanent damage behind it. The onset of the articular manifestations commonly follows the termination of the acute stage, when the temperature has fallen and the diarrhoea has abated. It is usually attended by a fresh elevation of temperature, but, speaking generally, the constitutional disturbance which attends its developments is slight. The largest joints are most commonly affected, and especially the knees; the elbows and ankles are frequently attacked, and sometimes the smaller joints also. The skin over the inflamed joints is not reddened, and there is little local heat or spontaneous pain. Movement of the joints causes pain, and there is tenderness on pressure, especially over the insertion of ligaments. There may be synovial effusion, sometimes of an extreme degree, especially in the knees. Salle describes the fluid removed by puncture as clear, yellowish in tint, and stringy, and as occasionally purulent. The arthritis usually subsides in a week or ten days, leaving behind it some temporary stiffness, but in most cases no permanent damage. Sometimes the arthritis is more persistent, especially when the smaller joints are affected, and Dewevre has seen it last as long as seven months. There is little or no tendency to relapse. It is rare for chronic and crippling articular lesions to follow dysenteric arthritis. As in gonorrhoeal rheumatism, neuralgiae, such as sciatica, and conjunctivitis may accompany the lesions of the joints.

Treatment.—Major Beveridge obtained the best results with quinine in five-grain doses. Salle recommends rest in cases without effusion, and the application of iodine or of *pointes de feu*. In cases with effusion, puncture and compression of the joints should be employed.

Syphilitic Arthritis.—Several distinct articular lesions may be met with as manifestations of syphilitis. These have been studied by Fournier and his pupils in France, and in this country by Prof. Howard Marsh, Messrs. Jonathan Hutchinson junior, Clutton, and others. A valuable general description of these forms of arthritis was published by Morestin in 1901.

The articular lesions differ in character according as they appear in the earlier or later stages of syphilis. Articular pains, apart from any objective signs, are not uncommon in the *early secondary stage*, and occasionally several of the larger joints, and especially the knees, are attacked by a subacute arthritis. The affected joints may be slightly swollen, and may

shew slight effusion. The skin over them may be of natural tint or somewhat reddened. The bodily temperature is little if at all raised. The pain may be acute, especially at night, and the affection resembles in many respects an attack of gonorrhoeal rheumatism of moderate severity. Much less frequently one or two joints, usually the knees, are the seats of an indolent hydrarthrosis, whereas all other signs of inflammation are wanting, and there may be so little pain that the condition is overlooked. These forms of arthritis yield to antisyphilitic treatment, the subacute arthritis readily, the painless hydrarthrosis less satisfactorily.

The articular lesions of the *tertiary stage* are of several kinds. In some cases the synovial membrane and the periarticular structures are the seats of gummatous infiltration, and there may be some effusion into the cavity of the joint, whereas the bones shew no signs of disease. In a form spoken of by Mr. Jonathan Hutchinson junior as chondro-arthritis, there is thickening of the synovial membrane and conspicuous hypertrophy of its fringes. The articular cartilages exhibit pits, which may extend down to the bones, and are lined with fibrous tissue. In another class of cases the affection of a joint is secondary to disease of the adjacent periosteum and bones, the ends of the bone are enlarged, and the hypertrophy extends to the diaphyses. At the same time there is effusion into the synovial cavity.

In *congenital syphilis* also the joints may be implicated. They may be attacked in connexion with the syphilitic epiphysitis of infants, and suppuration may even occur in them. Mr. Clutton has described a chronic hydrarthrosis, usually of the knees, in older children the victims of inherited syphilis; in the majority of his cases there was interstitial keratitis. Dr. Still is inclined to believe that the condition of the joints met with in children, which closely resembles the osteo-arthritis of later life, with osteophyte formation and nodes like those of Heberden, is of syphilitic origin; and Morestin speaks of bizarre thickening and outgrowths of the epiphyses as results of the inherited disease.

Pneumococcal Arthritis.—It has long been known that acute arthritis is an occasional though rare complication of pneumonia; but our more exact knowledge of such lesions, and the recognition that the pneumococcus produces morbid changes in many parts of the body besides the lungs, date from 1888 and the researches of Weichselbaum. Le Roux's monograph, published in 1899, laid the foundation of a more complete knowledge of pneumococcal arthritis in all its aspects, and Dr. E. J. Cave was the first British author to direct attention to the subject, in 1901. Since then this form of arthritis has excited much interest, and valuable contributions to its study have been made by Herrick, Dudgeon and Branson, Raw, Pasteur and Courtauld, Herzog, and others.

In adults this lesion occurs in direct association with pneumonia in the great majority of cases. It may precede the development of the

pulmonary lesions, but more frequently comes on during convalescence. In some instances it is merely an incident in the course of pneumococcal septicaemia, and may be associated with pneumococcal endocarditis. In children it may occur apart from any pulmonary troubles; and Mr. Dudgeon and Dr. Branson assign to the pneumococcus the chief part in the production of the acute suppurative arthritis of infants, and are supported in this opinion by Herzog's investigations. Even in later life it may occur as a primary lesion, as Dr. W. Pasteur and others have shewn, and in some cases it may be associated with other minor pneumococcal troubles such as otitis media.

Like other forms of arthritis due to pyogenetic organisms, pneumococcal arthritis is not always suppurative. The fluid in the synovial cavity may be creamy pus, sero-purulent, or even serous. Even in the same case suppuration may occur in one joint and not in another. The inflammatory changes may be confined to the synovial membrane, or may lead to erosion of the cartilages, and may invade the bones and periarticular structures.

In its *clinical aspects* pneumococcal arthritis resembles other varieties of acute inflammation of joints, and especially those of pyaemic origin. The swelling may be almost entirely due to synovial effusion, or may invade the periarticular structures. The skin covering the joint may be reddened, and there may be widespread oedema in its neighbourhood. Pain may be acute and severe or comparatively slight, and there is usually conspicuous tenderness when the joint is handled. The large joints are most often attacked, and the knees more often than any others. Of the smaller joints the sterno-clavicular would appear to be seats of election. Not uncommonly the arthritis is confined to a single joint. An injury old or recent seems to be, in not a few instances, the determining cause of the invasion of a particular joint, a feature which the pneumococcal shares with other forms of arthritis, including gout. The constitutional disturbance which attends the arthritis is chiefly dependent upon other circumstances. In cases with pneumococcal septicaemia it is, of course, intense; but when a single joint is attacked during convalescence from pneumonia, the onset may be marked by but little elevation of temperature or acceleration of the pulse.

Prognosis and Treatment. — Among cases in which pneumococcal arthritis occurs the death-rate is high. Thus, among 52 cases collected by Herrick no less than 34 were fatal. The treatment is mainly surgical. In suppurative cases incision and drainage of the joint is indicated, and has frequently been attended with excellent results. In some cases in which the effusion proved to be serous or semi-purulent, withdrawal of the fluid by puncture has sufficed to bring about a cure.

In cases of pneumococcal septicaemia, in which the arthritis is a minor incident, treatment of the joints is not likely to have any real influence on the result.

The accounts referred to above deal with cases in which the nature of the arthritis has been conclusively proved by bacteriological methods:

but, as Mauclaire has shewn, a suppurative arthritis complicating pneumonia is not necessarily due to invasion of the joints by the pneumococcus, nor is it possible to arrive at a definite diagnosis of pneumococcal arthritis on clinical grounds alone.

Some other forms of Purulent Arthritis.—The part played by the ordinary pyogenetic streptococci and staphylococci in causing pyaemic arthritis is well recognised and calls for no special description, but even these organisms may give rise to an arthritis which is not suppurative; indeed Singer and others believe that rheumatic fever is merely an attenuated pyaemia in the causation of which such micro-organisms play the chief part (*vide* Vol. II. Part I. p. 615). Again, as Max Schüller has clearly shewn, even in forms of secondary arthritis, in which the specific bacterium of the primary disease is found in the joint, micrococci are not infrequently present in preponderating numbers.

The meningococcus or *Diplococcus intracellularis* of Weichselbaum has also been shewn to be present in joints which are the seats of arthritis arising in the course of cerebrospinal meningitis, and of that attenuated form of the same disease which occurs sporadically as the posterior basal meningitis of young children.

Rhumatisme Tuberculeux.—In 1900 Poncet advanced the view that the articular lesions of tuberculosis were by no means confined to the more familiar and destructive lesions, the *tumeur blanche* of French authors. He maintained that, like other zymotic diseases, tuberculosis occasionally gives rise to a pseudo-rheumatic affection comparable to the gonorrhoeal and dysenteric varieties, and described both articular and abarticular manifestations of this form of pseudo-rheumatism. In some cases an arthralgia may, according to Poncet, be the sole manifestations in the joints, in others there is a synovitis which may be compared to serous pleurisy, and which in its clinical features and transitory nature may simulate true rheumatism, or rheumatoid arthritis. In not a few cases the joints recover entirely, in others the ordinary destructive tuberculous lesions develop, and in others again bony ankylosis results, as in the variety of spondylitis deformans called by Pierre Marie spondylose rhizomélique (see p. 41), of which Poncet believes tuberculosis to be probably one of the causes.

This question of tuberculous pseudo-rheumatism has since attracted much attention from French physicians, and has been discussed in various societies and journals. A study of the literature of the subject leads to the conclusion that not a little of the evidence brought forward is far from cogent, and that many of the cases adduced in support of the above thesis are capable of other interpretations. In some instances an acute or subacute arthritic attack, followed by complete recovery, has preceded, at a considerable interval, the onset of unequivocal manifestations of tuberculosis. In others the only evidence of a tuberculous origin is a

reaction to tuberculin, which, however convincing of the presence of a tuberculous focus somewhere in the body, can hardly be regarded as proving the tuberculous nature of the articular lesions. There are, however, cases in which the evidence available is far stronger, especially one recorded by Griffon. The patient, a man, aged 38, developed an arthritic attack with clinical features closely resembling those of gonorrhoeal rheumatism, in which many joints were implicated, including the sterno-clavicular and temporo-maxillary articulations. The joints most affected were one ankle and knee, of which the latter was the seat of a considerable effusion. Gonorrhoea could be excluded, and in default of any other diagnosis the possibility of tuberculosis was thought of, although there were no other manifestations of that disease in the lungs nor elsewhere. Cytological investigation of some fluid withdrawn from the knee shewed abundance of lymphocytes, as is the case in serous pleuritic fluid, whereas in true rheumatism and gonorrhoeal arthritis polymorphonuclear leucocytes preponderate. This evidence of a tuberculous origin of the arthritis received important support from the production of tuberculosis in guinea-pigs injected with the synovial fluid. Long before the guinea-pigs were killed the patient had made a complete recovery. It remains for future investigation to shew whether cases of this kind are at all common, and in how many of the cases regarded as examples of tuberculous rheumatism on clinical grounds and in which the lesions have developed in direct association with visceral tuberculosis, such evidences of a tuberculous nature of the arthritis are to be obtained.

Other varieties of infective Arthritis.—The forms of infective arthritis mentioned in the preceding sections by no means exhaust the list of such affections, for there are few zymotic diseases in connexion with which inflammation of joints has not been described as an occasional accident. Thus, arthritis has been observed in connexion with variola, measles, mumps, diphtheria, glanders, and erysipelas. In enteric fever it is not a very rare complication. Of the articular lesions of scarlatina, one class at least has a peculiar interest, because of its very close resemblances to true acute or subacute rheumatism. The resemblance is the more striking because it is apt to bring in its train some manifestations which are usually regarded as rheumatic in a more strict sense, such as chorea and even subcutaneous nodules. It is, indeed, difficult to believe that in some at least of the cases in which such troubles appeared during the period of desquamation, we have not to deal with true rheumatism complicating or following scarlatina.

In not a few of the above diseases the articular lesions are in some cases of a transitory and pseudo-rheumatic character, whereas in others they end in suppuration. For descriptions of these varieties of arthritis, in so far as they possess any clinical importance, the reader is referred to the articles dealing with the several diseases referred to.

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A. E. G.

INTERMITTENT HYDRARTHROSIS

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It is difficult to determine the place to be assigned to the bizarre phenomenon known as intermittent hydrarthrosis in any account of the maladies which implicate the joints, and whether it should be regarded as a disease *sui generis* or as a symptom only.

It consists essentially in a periodic effusion into the synovial sac, and in most instances recurs with almost mathematical regularity. In one class of cases this appears as an isolated or almost isolated morbid event, and between the attacks the affected joints return to their normal condition. In the cases of a second class the periodic effusion occurs in joints which are the seats of disease of one kind or another, and can then only be regarded as an unusual symptom. The division between these two classes of cases is not very sharply defined, and in some instances the difficulty of classification is by no means slight.

Perrin first described a case of the kind in 1845, and two characteristic examples were recorded by Grandidier in 1851. The first British cases were described by Moore in 1867. Of recent years the condition has figured more largely in medical literature, and important monographs upon the subject were written by Benda in 1900, by Linberger in 1901, and by Schlesinger in 1903.

Etiology.—Intermittent hydrarthrosis is met with in both sexes, but females cases somewhat preponderate. The great majority of first attacks occur between the ages of 10 and 40 years. In female cases the maximum incidence is at an earlier age than among males. In one case only, recorded by Blanc, has the occurrence of the condition in parent and child been observed.

In the primary cases the first attack has sometimes followed an injury. The attacks have occasionally been more severe when they coincided with a menstrual period. In some instances a relation to the menses has been suspected, and in one with a fortnight's cycle the alternate attacks preceded the menstrual periods. In some cases the attacks have ceased during pregnancy, but this does not always happen.

The symptomatic cases have been in examples of rheumatoid arthritis, gonorrhoeal rheumatism, and osteomyelitis due to a staphylococcus. Two cases that came under my observation presented many points of resemblance, and cannot be matched among the recorded examples. Both patients were men, and in both a characteristic intermittent hydrarthrosis of both knees, with accurately recurring attacks, appeared after gonorrhoeal rheumatism and continued for long periods, the knees shewing but little sign of disease in the intervals. Both

patients subsequently developed a crippling arthritis of almost all their joints, not to be distinguished from that known as rheumatoid arthritis.

Intermittent hydrarthrosis has been met with in association with nervous disorders of various kinds, such as epilepsy, migraine, and neuralgiae. Exophthalmic goitre has accompanied or has alternated with the joint affection in more than one instance, and swellings of the nature of giant urticaria, and recurrent urticarial eruptions have also been observed in a few cases. Another occasional symptom must be mentioned, to which Schlesinger has called special attention, viz. polyuria and dysuria at the time of the attacks.

Pathogeny.—Various hypotheses have been advanced to explain this remarkable affection. Malaria has been invoked, but the length of the cycle is so unequal in different cases that, even if it were possible to assume that all the patients had been infected with malaria, the periodicity could not be attributed to stages in the life-history of a parasite. It is clearly an idiosyncrasy of the patient rather than of the disease. The same objection applies to the suggestion that the phenomenon is due to a specific bacterium. As a matter of fact fluid withdrawn from such joints has almost always proved to be sterile, but experience teaches that in joint lesions due to bacterial invasion, the organisms are more likely to be found in the solid tissues of the joints than in the synovial fluid. Seeing, moreover, that intermittent hydrarthrosis may occur symptomatically in the course of various kinds of arthritis it would not be surprising if micro-organisms were more often found.

The view which has gained most acceptance is that which associates intermittent hydrarthrosis with acute circumscribed oedema, often styled angioneurotic oedema. There are good reasons for thinking that such a transient arthritis as that of rheumatic fever has a close kinship with such inflammatory forms of oedema as are met with in the erythemas and urticaria, and the frequency of their clinical association strengthens this belief; but one cannot help feeling that some writers have laid undue stress upon the presence, in one or two recorded cases of the articular trouble, of symptoms which appear to be frequently associated with acute circumscribed oedema. It is true that periodicity is sometimes noted in this latter affection, and Dr. Jamieson specially insisted upon this feature, which was presented in a marked degree in a case under his observation of a woman crippled by rheumatoid arthritis who developed circumscribed oedema of the eyelids and lips, usually on the same day in successive weeks. Indeed this periodicity of circumscribed oedema resembles more closely that of intermittent hydrarthrosis than does that of any other phenomenon that can be quoted. It is not mentioned at all, however, in many of the published descriptions of circumscribed oedema, whereas, in the case of the joint affection it is the salient and most surprising feature. We know so little of the causation of acute circumscribed oedema, that the recognition of its kinship with intermittent hydrarthrosis throws but little light

upon the pathology of the latter, and gives no clue to the factor determining its strange periodicity; the meaning of which remains as obscure as ever.

Taking all the known facts into consideration it seems safer, on the whole, to regard intermittent hydrarthrosis as a symptom which is occasionally manifested in the course of articular diseases of various kinds, and especially when such maladies affect the knees, than as a disease *sui generis* which may occasionally attack joints which are already the seats of lesions of other kinds.

Clinical Features.—In a characteristic case of the primary group the onset of the individual attack is sudden and without warning, save that which is afforded by the patient's experience of the periodicity of his trouble. A sensation of tension in the affected joint, usually a knee, is quickly followed by swelling due to synovial effusion, which may be slight or excessive. As a rule the skin over the affected joint is normal in appearance, and does not convey the impression of local heat to the observer's hand. Even at the height of the attack a surface thermometer applied to the joint may not detect any local elevation of temperature. On the other hand, cases are recorded in which redness of the skin and local heat have been present. After a period which is usually about three or four days the local phenomena quiet down, and the joint resumes its normal condition until the onset of the next attack. When the attacks have been very often repeated, the swelling may not completely subside in the intervals, some synovial crackle may persist, and even some degree of arthritic muscular atrophy may result. It should be mentioned that Linberger, as the result of careful measurements, arrived at the conclusion that the onset of the swelling was really gradual, and not so sudden as it appears to the patient to be. Pain is not a prominent feature in most cases, in some it is practically absent, and the patient merely experiences inconvenience from the effusion; in others again the amount of pain is considerable, and it is not always limited to the immediate neighbourhood of the affected joint or joints. The bodily temperature is seldom raised, and often there are no constitutional symptoms. In a few cases headache, gastric disturbances, some premonitory malaise, lassitude, or sensation of weight in the limbs have preceded the onset of the disease, or of each successive attack.

The length of the cycle varies in different cases, but usually remains constant or nearly so for each individual. Sometimes, however, the attacks are prone to anticipate and sometimes to postpone their usual appearance. Thus, in a case under my observation it became prolonged by about two days in the course of eight months. Benda, from the comparison of recorded cases, concludes that the commonest period is eight to eleven days, from the beginning of one attack to that of the next, and that periods of seven to nine days stand next in frequency, but in exceptional cases the cycle is much shorter, and in some has been as long as several months. One point stands out clearly, viz. that

the duration of the cycle is a peculiarity of the individual case, and is not controlled by any general law. The duration of the active stage also tends to be constant for the individual cases, but, as Benda further shews, there is no definite relation between the duration of the active stage and the length of the cycle. In the cases which he analysed the extreme limits of the active period were one day and twenty-one days respectively, but in about two-thirds of all the cases the duration was between two and eight days. Three days was the commonest duration of the swelling. It is difficult to convey, on paper, a just impression of the remarkable punctuality of the returns in many cases; not a few patients can foretell to a day and even to an hour the return of the symptoms, and are able to make their arrangements accordingly.

The cycle of events may repeat itself for months or even for many years, or a series of attacks may be followed by a period of immunity. The cessation of the attacks during pregnancy has been observed in a sufficient number of instances to exclude all possibility of accidental association, but occasionally the intermission has no obvious cause, and occasionally series of attacks may occur at corresponding periods of successive years. After such an intermission the old cycle may be resumed or a fresh period may become established.

The special liability of the knees is well shewn in the following table, quoted from Schlesinger's monograph, in which the primary and symptomatic cases are grouped separately. This table was compiled from recorded cases and those which Schlesinger had himself observed.

Seat of the Hydrarthrosis.	Primary cases.	Symptomatic cases.
One knee only	22	4
Both knees	18	2
One or both knees and other joints	12	4
Other joints alone	1	1
	53	11

Prognosis is very uncertain. In some cases the attacks have ceased spontaneously, or as the result of treatment. Others have resisted all therapeutic measures, and the cycle has been repeated without interruption for many years.

Treatment.—Of drugs, arsenic has the best record, but has sometimes proved useless. In a case seen by the writer a steady and almost uninterrupted recovery dated from the time that arsenic was tried, and threatenings of relapse were repeatedly averted by the same means. Quinine has proved very useful in some cases, valueless in others. Electrical treatment has been successful in some cases, and local surgical measures, for example, injection into the joint of irritant substances, such as iodine, have arrested the attacks in a few cases. However, whilst such

local measures have proved effectual in some cases, the recovery of one knee has occasionally been followed by the transference of the periodic affection to the other knee, previously sound. It is probably best in any case to commence by giving a fair trial to treatment with arsenic, and if this fail to resort to other measures. It may be mentioned that no encouraging results have been obtained by salicylic treatment. Complete rest appears to have no influence upon the recurrence of the attacks, nor does strapping or bandaging arrest the effusion, which indeed necessitates the removal of such applications.

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PULMONARY OSTEO-ARTHROPATHY

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Synonyms. — *Ostéo-arthropathie hypertrophiante pneumique* (Marie); *Sekundäre hyperplastische Ostitis* (J. Arnold) (2); *Toxigene Osteo-periostitis ossificans* (Sternberg); *Secondary hypertrophic osteo-arthropathy* (Massalongo); *Hyperplastic osteo-arthritis* (F. Rufenacht Walters); *Tuberculous Polyarthritis* (W. Thorburn); *Marie's sign-group.*

Since the publication of Pierre Marie's contribution on "Ostéo-arthropathie hypertrophiante pneumique" in 1890, various other titles have been suggested as more correct descriptions of the clinical and pathological features of this condition. Bamberger (4) indeed anticipated Marie's generalisation in a demonstration of two cases of this sign-group; and subsequently (5) described the clinical and pathological changes under the title "Knochenveränderungen bei chronischen Lungen- und Herzkrankheiten." Since pulmonary disease is not invariably present, and also for the sake of brevity, "pulmonary hypertrophic osteo-arthropathy" may be conveniently spoken of as Marie's sign-group.

Subsequent investigations, stimulated by the work of Marie and Bamberger, have considerably modified the details of Marie's first description; the affection may now be considered to be a group of

morphological changes consisting (1) of clubbing of the fingers and toes, to which none of the various synonyms draws attention; and (2) of certain changes in the ends of the long bones, more especially in the wrist- and ankle-joints, and of the cartilages and synovial membranes in these and occasionally in other joints. These changes are secondary to a morbid process in some other part of the body, although in a few cases it has been impossible to establish the nature or even the existence of the primary disease. It is obvious that the problems connected with the subject must be considered first with regard to the familiar "clubbing" of the digits, and secondly with regard to the much rarer and no less obscure condition of osteo-arthropathy.

I. Clubbing of the Digits.—Synonyms: *Les doigts hippocratiques, Hippocratisme* (Pigeaux, Trousseau); *les doigts en baquette de tambour; les doigts en tête de perroquet; Trommel-schlägelfinger und -zehen.*

Definition.—The name "clubbing" of the fingers, or of the toes, denotes the appearance of the digits when the last phalanx is swollen and globular, and the nails become enlarged and much more convex than normal both in their longitudinal and transverse diameters.

This deformity may or may not be associated with some cyanosis, and usually affects all the digits of the hands and feet, though often unequally. The thumbs and index-fingers are said by Trousseau to be affected earliest; first in the right hand and then in the left. The skin about the lunula may be highly polished, and it is noteworthy that this change of the skin and slight degrees of swelling of the last phalanx may precede any marked increase of curvature of the nails. According to Erich Ebstein, the earlier writers, Hippocrates and Aretaeus, confined their attention to the increased curvature of the nails; it was Caelius Aurelianus who first drew attention to the drumstick or clubbed appearance of the digits. Possibly the alteration of the nails chiefly appealed to these observers, because this change was then attributed to wasting of the soft tissues. The name "clubbed" or "drumstick" fingers is not only more graphic, but apparently more correct historically than "hippocratic fingers," the name generally used by French writers.

The alteration in shape of the fingers and toes is unaccompanied by any disturbance of sensation such as anaesthesia, paraesthesia, or hyperalgesia. Notwithstanding a prevailing opinion to the contrary, there does not appear to be any sharp difference in the appearance of the clubbed digits when these occur alone or as a part of Marie's sign-group, other than would depend upon the added changes which in the latter case take place in the bones of the first, second, and even in the third phalanges, and in the interphalangeal joints.

Etiology.—As a curiosity it has been reported in healthy subjects (West, Janeway).

(*a*) *Diseases of the Lungs and Pleurae.*—The most exquisite examples of clubbing are seen in bronchiectasis, but it is not constantly present in this disease. In 57 cases of bronchiectasis among out-patients at the Brompton Hospital, 33 being females and 24 males, 20 cases only

shewed the classical signs above referred to in a marked degree; in 18 others it was obvious but not very striking, and in the remainder there was no clubbing, nor even increase of the convexity of the nails or glossiness of the skin near the lunula. The frequency of clubbing in pulmonary tuberculosis has been variously estimated. Dr. J. E. Pollock noted the change in 29 per cent of the male and 23 per cent of the female cases. In 600 cases of tuberculosis of the lungs with tubercle bacilli in the sputum at the Brompton Hospital, clubbing was met with in 69·6 per cent of the male and in 66·4 per cent of the female cases. Well-marked clubbing was met with in 20 of the cases only, obvious clubbing in 47, slight changes in 345; in the remaining 188 cases no clubbing nor any sign of nutritional change of the finger-ends was noticed. Bilateral clubbing of the digits may occur in abscess of the lung, in empyema, especially when ill-drained, in emphysema (Percy Kidd), and in chronic pneumonia (Pye-Smith).

(*b*) *Diseases of the Heart.*—Clubbing also occurs in a marked degree in congenital heart disease, more especially in cases with pulmonary stenosis and an incomplete interventricular septum, but is not usually present in the cases in which the ductus arteriosus is patent and dilated. It may also occur in acquired heart disease, but this is rather exceptional, at any rate in the fully developed form.

(*c*) *Diseases of other Organs.*—Bilateral clubbing has also been met with in diseases of organs other than the lungs. It has been recorded in chronic jaundice and biliary cirrhosis (Gilbert and Fournier, Gilbert and Lereboullet, F. Taylor, Roger-Smith), in cicatricial pyloric obstruction (Dennig), and in obstructive jaundice (Rolleston). Stokvis and subsequently Van den Bergh have noted clubbing in the condition named by Stokvis "autotoxic enterogenous cyanosis." Clubbing is a constant feature in Marie's sign-complex. Janeway refers to clubbing in isolated cases of purpura (Mangelsdorf), pyelonephrosis and cystitis, carcinoma of the oesophagus, leprosy, rickets, and malarial cachexia; it has been noted, in association with perforating ulcer of the foot and neuritis, in hypertrophic biliary cirrhosis, and in polycythaemia with enlarged spleen.

Unilateral Clubbing, which has been described in association with aneurysms of the thoracic aorta and its branches by Canton, J. W. Ogle, Sir Thomas Smith, Prof. Osler (2 cases), Béclère (6), and Groedel, is an extremely interesting condition.

Clubbing, though usually very slow in its progress, may appear within a week (Saundby, quoted by Ebstein), or a fortnight (S. West). The disappearance of clubbing is well known after the cure of various associated conditions such as empyemas (S. West, Moizard, Orillard). Krüger states that the clubbing disappeared after the relief of cicatricial pyloric obstruction; in Dr. West's case the clubbing was seen to disappear in three months. In Sir T. Smith's case the clubbing became much less marked after ligature of the subclavian artery, although the patient only lived about 20 days after the operation.

Morbid Anatomy.—Different observers have described various changes

in the soft parts: fibrous thickening of the rete mucosum (Buhl); dilatation of the capillary loops under the nail, enlargement of the interpapillary processes without any other alteration of the skin (Freytag, quoted by H. E. Symes-Thompson); and increase of the connective tissues (Liebermeister). Dr. Norman Moore does not confirm this last observation, for he found that pressure would reduce the swollen finger to the normal size. In pure clubbing of the digits, according to radiographic and other observations, there is no bony alteration (Dennig, Litten, Hirschfeld). The increase of bone in the last phalanx, observed by Groedel and others, is possibly merely a manifestation of the bony change met with in Marie's sign-group. Although Dr. Norman Moore's observation suggests that clubbing is due to oedema alone, it is generally agreed that oedema plays no part in the development of the deformity.

Pathogenesis.—Whatever the origin of the change, there is little doubt, judging from the rather scanty observations, that the clubbing is due partly to vascular turgescence and partly to hyperplasia of the soft parts. The mechanical effect of congestion has been advanced to explain the clubbing in cardiac disease, and even for the clubbing met with in disease of the lung, although it is not quite clear why the temporary pressure of an empyema on the lung should produce clubbing. The advocates of the mechanical origin maintain that those diseases of the lung and even of the liver that are associated with clubbing are instrumental in that they hamper the action of the diaphragm and in this way produce stagnation of the general circulation. More recently Bezançon and de Jong suggest that clubbing is the result of dilatation of the right heart, though, as Bezançon admits, this will not explain unilateral clubbing. Béclère (7) has modified the toxic conception of the causation of clubbing: instead of adopting the view that the diseases of the lung, whether primary or secondary to heart disease, or of such organs as the liver, produce a toxin which causes clubbing, he considers that venous blood naturally contains substances which provoke changes in the fingers; and that if during its passage through the lungs this substance be not removed from the blood clubbing results. This mechanico-toxic hypothesis is most ingenious, and helps to explain not only bilateral but also unilateral clubbing. Finally, by Sahli, the condition has been regarded as neuro-trophic, but this conception has met with little support; moreover, it is noteworthy that in a case of Marie's sign-group, Dr. Farquhar Buzzard did not find any causal change in the peripheral or central nervous system.

Diagnosis.—There can be little difficulty in the diagnosis of marked clubbing except when injury or whitlow has deformed the end of the fingers, or, as in the case of sailmakers and bootmakers, when the thumb has become expanded at the end.

Prognosis.—With the cure of the primary disease, such as empyema or aneurysm, the clubbing disappears. As a means of prognosis, however, the presence or absence of clubbing is valueless.

II. **Marie's Sign-group.**—*Definition.*—As already stated, this group of signs consists of clubbing combined with various changes in the long

bones and in the joints, the latter conditions being frequently spoken of as "osteo-arthropathy."[1] Bamberger's pathological investigations and recent observations by means of *x*-rays have brought into full prominence the changes underlying this curious clinical condition. Besides the clubbing of the digits, the joints of the wrist, ankle, carpus, tarsus, and even the interphalangeal joints, and the joints of the elbows and knees, are affected, and the distal ends or whole shafts of the radius, ulna, tibia, fibula, humerus, and femur become expanded, thus producing in great measure the characteristic swellings of the extremities. The patella and, in extreme cases, all the bones of the body may be enlarged. It appears that thickening of the shafts of the metacarpal, metatarsal bones, and phalanges may be found; and, as shewn by Chatin and Cade and Dr. Donald G. Hall, there may be osteophytes on the terminal phalanges. Pain in the affected joints and bones is a variable feature, indeed the bone changes may be quite painless. Some patients are greatly crippled, being unable to perform fine movements, such as writing, or even to walk about.

Etiology.—(*a*) *Diseases of the Lungs or Pleurae.*—As in clubbing of the digits, so here pulmonary disease is the most frequently associated condition; this was so in 43 out of 55 cases collected by Thayer, in 65 out of Janeway's 93 cases, and in 68 of Wynn's 100 cases. Teleky's and Janeway's collections of cases shew that the sign-group has been seen in bronchiectasis, chronic empyema, pulmonary tuberculosis, especially with excavation, abscess of the lung, pneumonia, pleurisy, and malignant disease of the lung and pleura. By far the larger number of cases occur in association with the first three of these causes, but it must be admitted that the sign-group is extremely rare even in association with pulmonary lesions.

(*b*) *Diseases of the Heart.*—Although clubbing of the digits is comparatively common in congenital and even in acquired heart disease, Marie's sign-group has been very rarely recorded in the latter condition, and in one instance only in connexion with congenital heart-disease (Batty Shaw and Higham Cooper). In Bamberger's eleventh case—one of aortic disease, though not diagnosed as one of osteo-arthropathy—a definite osteophytic growth was found after death at the lower end of the left radius. Mr. Thorburn described Marie's sign-group in a boy with mitral stenosis and no lung disease.

(*c*) *Disease of the Subclavian Artery.*—Just as unilateral simple clubbing has served to crystallise a new view of the pathogenesis of this condition, so also Berent's remarkable case of unilateral Marie's sign-group, dependent upon an aneurysm of the left subclavian artery, has had a similar effect. In the left upper extremity there was clubbing of

[1] Some of the features originally described by Marie have now been excluded, such as the changes in the face. When it is remembered that one of the cases originally described by Marie as an example of pulmonary hypertrophic osteo-arthropathy was subsequently proved to be a case of acromegaly, it may be well understood that changes have had to be made not only in the name introduced by Marie, but also in the actual description of the disorder.

the fingers and oedema of the forearm, and, as shewn by *x*-rays, expansion of the lower ends of the radius and ulna with a generalised rarefaction of bone such as was described by Teleky, Kienböck, and Sudeck. In Groedel's case of unilateral clubbing due to an aneurysm of the arch of the aorta implicating the left subclavian artery, *x*-rays shewed bony enlargement confined to the terminal phalanges.

(*d*) *Other Diseases in which Marie's Sign-group has been stated to occur.*—The occurrence of Marie's sign-group has been reported in the following diseases:—Diarrhoea (32), dysentery (65), influenza (50), syphilis (54), chronic jaundice (43), alcoholism (61), inoperable carcinoma of the stomach (33), biliary cirrhosis (14, 21, 22, 68), chronic nephritis (14), secondary pyelonephritis (18, 37). Marie's sign-group has been observed in some diseases of the nervous system by various writers, for example, in syringomyelia (27, 34, 36, 55). It has been suggested that in Berent's and Groedel's cases neuritis was the cause of Marie's sign-group, the neuritis being either toxic and due to pulmonary disease or the result of direct pressure. Finally, in some cases no cause has been discovered (16, 20, 58, 61A, 69).

Morbid Anatomy.—The changes in the long bones are usually quite symmetrical, but may be more advanced on one side or in the lower extremities, or be present in one arm only; the long bones of the forearm and leg are more affected than the humerus or femur. In the sites of bony expansion the periosteum is more adherent than in health; besides enlargement of the bones due to proliferation of bone beneath the periosteum, rarefaction takes place in the compact tissue of the shafts of not only the large long bones, but also of the metacarpal and metatarsal bones and the phalanges; Chatin and Cade, Donald G. Hall, and Groedel have shewn that the terminal phalanges may undergo hyperplastic changes even with the formation of spicules of new bone. Messrs. Thorburn and Westmacott point out that the changes in the long bones are often very much more extensive than would be expected from clinical examination, and that even the whole length of the shaft may be thickened by the addition of new bone. There may be symmetrical erosions of the articular cartilages, and evidence of recent synovitis or thickening and villous changes in the synovial membrane. From an analysis of the results of 21 autopsies, Dr. H. E. Symes-Thompson finds that among the bones more rarely affected are the clavicles, ilia, ribs, scapulae, and vertebrae. Dr. Farquhar Buzzard's microscopical examination of a finger, and of the nervous system, of a case of Marie's sign-group shewed that there was an increase of the fat in the finger and that the peripheral nervous system was quite normal.

Pathogenesis.—There are two views of the relation of the causative agency or agencies of Marie's sign-group and of clubbed digits. (1) Since the osteo-arthropathic lesions of Marie's sign-group have never been found apart from clubbing of the digits, many writers regard the osteo-arthropathy as a further result of the same cause or causes responsible

for clubbing of the digits. In the absence of more extended and exact pathological examinations of the bones and joints in simple clubbing, it is conceivable that even in these cases the osteo-arthropathic changes have begun, though not recognisable at the bedside or even by *x*-rays. Accepting provisionally the unity of the two conditions, more knowledge is required as to why the osteo-arthropathy should be so very rare, whereas clubbing is comparatively common. The occurrence of unilateral osteo-arthropathy and clubbing strongly suggests that the clubbing and the osteo-arthropathy are due to one and the same cause or causes; and the hypothesis put forward by Béclère and supported by Groedel's observation—a combined mechanico-toxic explanation—would appear to be more satisfactory than one purely mechanical or purely toxic. The nature of the locally developed toxin which can provoke changes both in the soft parts and in the bones and joints, and the nature of the process by which such body or bodies are under normal conditions destroyed when the blood reaches the lungs, are unknown. The only experimental investigation bearing on the lung-toxins supposed to be concerned in the causation of Marie's sign-group is that carried out by Bamberger; he injected per rectum the fetid contents of bronchiectatic cavities into young rabbits during five to six weeks; the bones of these animals were found eventually, when compared with those of control animals, to be unaltered; although negative as to the existence in bronchiectatic or tuberculous vomicae of any toxic agent capable of producing such changes, these results do not militate against the mechanico-toxic view advanced by Béclère. Lastly, as regards a neuropathic hypothesis, Marie's sign-group has been described as a result of syringomyelia and neuritis, but there is no evidence of any such association in simple clubbing of the fingers. Hence it is clear that a neuropathic explanation cannot apply to both these conditions.

(2) The dualistic causation of simple clubbing and of Marie's sign-group has attracted considerable attention, and it is suggested that clubbing is due to mechanical congestion alone, and osteo-arthropathy to quite a different cause, such as some infective process, and so belongs to the same category as Bouchard's pseudo-rheumatism or Ord's osteo-arthritis—which are the outcome of the toxic effects of organisms present in chronic urethritis, vaginitis, and so forth. In favour of this it may be argued that in many cases of Marie's sign-group toxins have been produced, for example, in bronchiectatic cavities and pulmonary abscess. On the other hand, this hypothesis fails to explain unilateral osteo-arthropathy, and therefore must be abandoned, in spite of Dr. H. E. Symes-Thompson's interesting but isolated observation of acute joint-attacks which recurred several times in the course of bronchiectasis and then terminated after a large quantity of sputum was voided. The same objection applies to Mr. Thorburn's view that the osteo-arthropathy is the result of a low form of tuberculous infection, and to Mr. R. J. Godlee's suggestion that possibly syphilitic infection is responsible for the alteration on the ground that the changes

in the articular cartilage in syphilitic arthritis and in Marie's sign-group are identical. The same may be said for the view, suggested by the title of Gerhardt's paper, that the articular and bone changes are simply due to rheumatoid arthritis or, according to Massalongo, to rheumatism supervening on simple clubbing. Lastly, there remains the possibility that Marie's sign-group is due to two factors—(*a*) mechanical, responsible for the clubbing; and (*b*) neurotrophic, responsible for the osteo-arthropathy. The clinical associations, already referred to, of Marie's sign-group with syringomyelia and neuritis suggested this explanation, which has been recently supported by Berent and Bernhardt. Berent considers it highly probable that nerve disturbance takes a large share in the production of the osteo-arthropathy, and that congestion and toxins developed from suppurative foci are not the only factors. The microscopic evidence for this view is somewhat slender; Moebius found neuritis of the ulnar nerve and osteo-arthropathic changes in the neighbouring tissues in a case of bronchiectasis; Hirschfeld described in three cases a condition, which he called vasomotorial dermatohypertrophia, consisting of symmetrical enlargement of the hands and feet associated with a club-like deformity of the distal phalanges, intermittent oedema, and exacerbations of pain and tenderness of the nerve trunks. As already stated, however, Marie's sign-group may occur quite independently of pain, whether spontaneous or produced by pressure; and against Moebius' observation may be set the normal condition of the peripheral nervous system in Dr. F. Buzzard's case.

In the light of our present knowledge, the mechanico-toxic view of Béclère, incomplete as it is, provides the best explanation not only of Marie's sign-group, but also of clubbing of the digits, which develops before or at the same time as the osteo-arthropathy, never after, and moreover need not be succeeded by any osteo-arthropathy at all.

Diagnosis.—Following the modern definition of Marie's sign-group by virtue of the assistance of radiography, mistakes are now less liable to occur in the differentiation of acromegaly from this condition. So far as the limbs are concerned, it may be said that in acromegaly the hands and feet are not deformed but are merely much enlarged, and that there is no proliferation of the subperiosteal bone or rarefactive change in the compact tissue of the shafts of the long bones; in acromegaly there is no change in the joints but obvious deformity occurs in the face, especially in the lower jaw, whereas in Marie's sign-group there are, according to modern views, no changes in the face, nor is spinal curvature considered such a constant feature as formerly, and when present, occupies a more varying position than the more gradually produced deformity met with in acromegaly. Moreover, in a large proportion of cases of Marie's sign-group some primary cause for the deformity is present. Pain occurs in the affected parts, more commonly in Marie's sign-group than in acromegaly, although acroparaesthesiae are fairly common in the latter disorder; in acromegaly there is usually no great limitation of the movement of the limbs, whereas in Marie's

sign-group this may be extreme and very painful. Cases of combined acromegaly and Marie's sign-group have, however, been recorded by Thayer (64) and by Jolly. Confusion of Marie's sign-group with osteo-arthritis, rheumatoid arthritis, syphilitic or tuberculous arthritis, may, as in the case of acromegaly, be obviated by the presence of the characteristic clubbing of the digits in Marie's deformity.

Prognosis and Treatment.—As in simple clubbing of the fingers, the soft parts may return to their normal condition when the primary disorder is cured, but, as might be expected, the new bone persists, though possibly some of the arthritic phenomena, such as synovitis, may subside. When the affected joints are painful, the best remedy appears to be fixation.

H. Batty Shaw.

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In some of the above references, it will be found that the cases described as acromegaly are really examples of Marie's sign-complex, *e.g.* the reports of Gessler and of Redmond; Arnold (2) shewed that one case described by Marie as pulmonary hypertrophic osteo-arthropathy (Hagner) was really one of acromegaly. For a very complete account of Marie's sign-group the reader may refer to an article by Dr. Finley Alexander, *St. Barth. Hosp. Rep.*, 1906, xlii. 41.

H. B. S.

OSTEITIS DEFORMANS

Leontiasis Ossea

By Anthony Bowlby, C.M.G.

Osteitis Deformans was first described by Sir James Paget in the year 1877. Since that time many examples of the disease have been recorded; in 1901 Packard, Steele, and Kirkbridge collected sixty-seven authentic cases.

It may be defined as a very chronic inflammation of bone occurring in people past middle age, implicating many bones, and accompanied by a peculiar softening and bending of the osseous structure.

It should be noted that in osteitis deformans there is no implication of the general health and no affection of the viscera. The onset is quite insidious, and it is not until some definite deformity of a limb or a considerable enlargement of the head attracts attention that any notice is taken of the bony thickening.

Pathology.—The bones affected by this disease become gradually thickened by the deposit of new bone from the periosteum, and by the same process their normal outlines are slowly obliterated. By the exudation of inflammatory products within the bone the osseous structure is absorbed, rarefied, and softened; the spaces thus formed are filled with inflammatory exudation. The whole bone becomes greatly increased in circumference, and, on section, is seen to be much thickened. In some

cases the medullary canal is increased in size. In consequence of the softening which accompanies the inflammatory process the bones become bent, the normal curves being at first increased; and, after a time, fresh curvatures are established.

The skull is increased in thickness, the forehead becomes large and prominent, and the face, in consequence, appears to be too small for the cranium by which it is overshadowed. The clavicles become much curved and thickened. The thorax falls in on account of the yielding of the softened ribs, and the abdomen becomes prominent. The femurs curve chiefly outwards, and the tibias forwards. The humerus does not curve so much as most of the long bones, but the radius and ulna curve backwards. In consequence of the bending of the bones of the lower extremity and of the general posterior curvature which is often met with in the spine, the height of the patient is frequently diminished by several inches. The walk is tottering, and the support of a stick is often necessary. The shoulders fall forward over the chest, and the head protrudes in a very peculiar manner, as if too heavy for the cervical vertebrae. The chin tends to rest upon the sternum, and in order to look up, the patient thrusts it out so that the face is carried on a plane which is considerably anterior to that of the body. The course of the disease is slow, and usually extends over many years. A fatal termination may result from the difficulty of respiration caused by the softened thoracic wall, and malignant tumours seem to occur in patients affected with osteitis deformans more frequently than they do in most elderly people.

Indefinite lesions have been found post-mortem in the spinal cord (2).

Symptoms and Diagnosis.—Beyond the deformity already described and some aching pains in the extremities there are no noteworthy symptoms. The diagnosis from acromegaly is easily made if it be remembered that in acromegaly the facial bones and those of the hands and feet are chiefly affected, while in osteitis deformans the bones of the cranium and the long bones of the extremities are mainly affected. In a few cases the disease is limited to a single bone; I have seen it so limited for several years to the humerus, the femur, or the tibia.

Treatment.—No treatment is known to produce any healing effect on the softening and bending of the bones. In a few instances iodide of potassium has appeared to relieve the pains.

LEONTIASIS OSSEA is the name applied to an overgrowth or hyperostosis of all or some of the cranial or facial bones. The formation of new bone is very slow, and may produce no noticeable deformity for many years; and the change may begin at any period of life, having been observed in childhood in several instances. Both sexes are prone to attack; the causes are unknown. The suggestion that the disease is due to rickets is quite unsupported by the facts observed in most cases. There are no concomitant affections of other parts of the body, and any symptoms that ensue are the direct result of the pressure exercised by the new bone. The superior maxillae and the various bones entering

into the formation of the inner walls of the orbits are not infrequently affected alone, the cranial and lower jaw bones remaining free from disease. In other cases the whole of the cranial and facial bones are affected, but the cranial bones are but seldom attacked alone. In the process of growth the affected bones become greatly increased in density, and the open cancellous framework of the ethmoid becomes converted into dense, compact bone. All cavities, such as the antrum, the sphenoidal and ethmoidal sinuses, and the bony canals for the supra- and infra-orbital nerves, become obliterated and their contents destroyed. In addition, there is considerable formation of new bone on the surfaces, and consequent deformity of the features. The cavities of the orbit, the nose, and the mouth may be encroached upon, and proptosis or blindness may ensue upon the pressure exercised upon the eyeball and the optic nerve. The sense of smell may similarly be lost from occlusion of the nostrils or pressure on the olfactory nerve in the ethmoid bone. A single superior maxilla is sometimes alone affected by hyperostosis, and may present precisely the same appearances as a bone from a case of leontiasis. Such an affection of one upper jaw is an infinitely more common disease than is leontiasis ossea: the latter is indeed a very rare affection. The symptoms caused by leontiasis vary with the position and extent of the new bone. Pain is necessarily very common, and occasionally mastication and speech are interfered with. The deformity is sometimes very great. No treatment can be adopted with any hope of success; but occasionally, when the growth is a limited one, some benefit may result from resection of some portion of the affected bone.

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A. B.

MOLLITIES OSSIUM

MULTIPLE MYELOMA

By ANTHONY BOWLBY, C.M.G.

Mollities Ossium, or **Osteomalacia,** is a disease in which there is a gradual softening and subsequent bending of the bones, and in which spontaneous fractures are very apt to occur.

Etiology.—No definite cause for mollities ossium can be assigned; but it has been stated that the absorption of bone is due to an excess of lactic

acid in the blood, though this excess has not been clearly demonstrated. When occurring during pregnancy the disease has been attributed to the demands of the foetus for bone salts, but this supposition does not apply to the cases which occur independently of pregnancy. It has been suggested that the disease is of ovarian origin, and in support of this attention has been drawn to improvement or recovery after removal of the ovaries; it has been thought to be a trophoneurosis, and due to reflex irritation from the ovaries, or to the result of an abnormal internal secretion of the ovaries which decalcifies the bones. A serious objection to the ovarian hypothesis is the occurrence of the disease in males. It may be stated that in many cases some causes of nervous or mental depression appear to have been present; but how such antecedents could lead to or bring about the changes described is beyond our comprehension. Mollities ossium occurs with far greater frequency in women than in men. It usually begins between the ages of 25 and 45, and its onset is frequently associated with pregnancy. More rarely it begins about the age of puberty, or even in childhood.

Pathology.—An examination of the affected bones shews that they are much lighter than natural, and so soft that they may be indented with the fingers or bent by the hands. They are readily cut with a knife, and the section shews that the medullary canal is greatly increased in diameter, and has extended into the epiphyseal ends. In advanced cases the compact tissue is reduced to a mere shell. Microscopical examination reveals an absorption of the lime salts, beginning around the Haversian canals and the canaliculi, such as occurs when bone is placed in a solution of hydrochloric acid; thus, while the lime salts are dissolved the animal matrix remains. In this matrix changes ensue of a degenerative nature, which end in a complete gelatiniform degeneration, and the formation of a jelly-like mass. In this process of destruction there is no sign of inflammation; but the vessels, deprived of their normal support, very frequently permit of exudations of blood, and the osteoclasts are much increased in number. A microscopical examination of the medulla and of the decalcified bone shews many blood-cells in various stages of disintegration, with cells of various shapes and sizes and granules of oil, fat, and gelatinous matter.

Symptoms.—Before any definite changes are noticed in the bones so-called "rheumatic" pains usually occur, and these may be severe; at the same time the general condition is one of feebleness or debility. The next change to be noticed is a sense of weakness and insecurity in the lower extremities, so that the patient is unwilling to walk without support, and is unable to walk with freedom; the gait is often peculiar, and the steps are short and unsteady. After this it may be noticed that one or more of the bones are becoming curved, so that the stature of the patient is diminished; whilst in other cases nothing so definite is observed until a bone is fractured by some trivial accident, or until parturition is impeded by deformity of the pelvis.

In advanced cases the deformity may be very great, and it may affect

the whole skeleton. The spine, by a great increase of its normal curves, assumes somewhat of the shape of the letter S. The ribs are flattened or even bent inwards on the lateral aspects of the chest, whilst near to the sternum and close to the spine they are protruded or fractured. The sternum is usually pushed forwards, and is often fractured at the juncture of its several portions. The pelvis is most seriously affected, so that parturition may become impossible. The sacrum is pushed downwards by the weight of the body, whilst the acetabula are pressed inwards by the heads of the femurs; thus the pelvic brim assumes a trifoliate form, with the pubic symphysis thrust forward like a beak. The bones of the extremities are bent or fractured in various degrees, and the whole of the limbs may become quite flaccid and useless. The urine contains an excess of lime salts. Lipaemia was observed in two cases (Klotz).

The viscera are not affected except as a direct result of the osseous lesions; but, in consequence of the latter, pulmonary congestion and enfeebled respiration are of common occurrence, and an attack of bronchial catarrh may at any time prove rapidly fatal. It should be remembered that the course run by different cases varies very greatly in rate, and that while in some patients all the changes enumerated may follow each other in the course of a year or a little more, in others the affection is limited to the pelvis and sacrum, and appears to progress only during pregnancy, either remaining stationary at other times, or making hardly perceptible progress. Some patients thus live for many years after the onset of the disease, and do not become bedridden at any time.

Treatment.—There is no specific treatment for mollities ossium, but good food and a general tonic line of treatment are indicated. Fractures in the early stage of the disease generally unite with proper care, although they do so slowly.

MULTIPLE MYELOMA.—Synonyms: *Myelomatosis; Kahler's Disease; Myelopathic Albumosuria; General Lymphadenomatosis of Bones.*—This disease is characterised by softening of certain parts of the skeleton. It is due to a tumour-growth, and is mentioned here because the bony deformities to some extent simulate those of mollities ossium, although they characteristically affect very different bones. It is a rare condition, and has been specially described by Dr. Parkes Weber, who collected between thirty and forty cases in 1903. He summarises his description as follows:—"Multiple myeloma may be defined as a diffuse new growth, primarily involving the bone-marrow, especially that of the vertebrae, ribs, and sternum, and affecting males as or more often than females, and chiefly those past middle age. The disease nearly always remains limited to the osseous system, though by direct extension it may form localised outgrowths projecting from the bones. Owing to absorption of the hard osseous tissue the bones become softened or friable, and are easily broken. The vertebral column and sternum are sometimes much bent, and the spinal cord may be affected by pressure, due to the curvature of the spinal column or to new growth bulging into the spinal canal. Owing

to the destruction of bone-marrow the formation of blood is impaired, and anaemia and progressive cachexia occur." It generally proves fatal within one or two years. The growth is of a sarcomatous nature, and in many cases the cells resemble small or large lymphocytes.

In many of the patients the urine is found to contain a peculiar form of albumin, which has been described as the "Bence-Jones proteid." When the urine is acid this protein has the following characteristics. It coagulates at about 58° C., and it is more or less completely dissolved by further heating to the boiling-point, or by adding acetic acid to the boiling urine. It deposits after boiling if the urine is allowed to cool. In Dr. Bradshaw's case the Bence-Jones albumose was precipitated spontaneously from the urine.

Osteopsathyrosis.—This name has been given to a form of bone-softening which occurs quite independently of age or sex. Many bones may be affected, and fractures are of more frequent occurrence than mere bending, although both may be seen. In some of these cases there seems to be a deposit of the absorbed bone salts in the kidneys, and in some such cases large renal calculi have formed. This condition has been named "calcareous metastasis."

Neuropathic softening of bone is described in Vol. VI. p. 545 (1899).

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A. B.

RICKETS

By W. B. Cheadle, M.D., F.R.C.P., and F. J. Poynton, M.D., F.R.C.P.

Synonyms.—*Rhachitis*, *Rhachitismus*, *Morbus Anglicus* (*Englische Krankheit*).

Short Description.—Rickets is a general disease of early childhood affecting the nutrition and development of the whole of the growing organism. The most marked physical changes are seen in the bones.

Ossification, which is still in active process, is both retarded and perverted. The bones grow irregularly, and, remaining largely cartilaginous and soft, yield under traction and pressure, thus giving rise to various distortions and deformities. The evolution of the teeth is delayed, their structure is fragile, imperfect, and subject to early decay. This striking affection of the osseous skeleton has concentrated attention upon one special feature of the disease, and fostered too narrow a view of its pathology. Rickets has until recently been regarded as chiefly a disease of bones; it has been thus classed in the text-books, and its etiology and pathology considered almost entirely in this relation. The defect of growth and nutrition is, however, by no means limited to the bony framework of the body: all the chief structures are affected. The muscles are wasted and remarkably enfeebled, the ligaments relaxed. The mucous membranes exhibit an abnormal tendency to catarrh. The brain is functionally backward, while the nervous system shews increased reflex irritability, so that various forms of spasm and convulsion are readily excited. The blood is impoverished, its red corpuscles being diminished to such a degree that in some cases the anaemia is well marked. Lastly, in some cases there is fibroid enlargement of the liver and spleen.

History.—The name, primarily derived from the Old English verb *wrikken*, to wrest or twist awry, arose no doubt from the application of a term, commonly applied in the country to inanimate structures, to a disease the most striking characters of which are contortion and deformity. The ill-formed child with the soft and loose-jointed framework of its body bent and giving way was called rickety, just as a dilapidated table or chair was so styled, and the condition received the name of "The Rickets." Glisson, who first described the affection in the seventeenth century, proposed the conversion of the common term "Rickets" into "Rhachitis," partly on account of the similarity of the sound, partly as a derivation from the Greek ῥάχις, the spine, on the ground that the dorsal spine is one of the first parts attacked. This, rather than Trousseau's derivation from the Norman word *riquets*, appears to be the correct origin of the English name.

Rickets was recognised on the Continent soon after Glisson's description of it in England, whence it was supposed to have spread; but it has no doubt existed in Europe from early times as an accompaniment of civilisation, and it is now becoming common in the younger countries of America and Australia.

Etiology.—Rickets is the result of imperfect and perverted nutrition; so far pathologists are agreed: there is, however, some divergence of opinion as to the exact causes of this defect. Most of the hypotheses have been based solely upon a consideration of the morbid changes met with in the bones, as if this comprised the whole pathology of the disease. To trace the nature and source of these faults of ossification may supply the key to the nature of the general affection, but no pathological doctrine can be regarded as satisfactory which does not also explain the morbid

conditions of muscle and tendon, mucous membrane, and nervous system: these conditions are concurrent and constant, although they are less obvious and obtrusive than is the affection of the skeleton. The production of a rickety condition of bone by the application of Esmarch's bandage, as in the experiment of Kassowitz, does not produce the changes in the other tissues. The bones are certainly soft and they give way under pressure, but the ligaments are also soft and lax, and give way under pressure likewise; the muscles are atrophic and enfeebled; the mucous membranes catarrhal; the reflex mechanism of the nervous system is hyperexcitable and unstable. It is difficult to understand how the bone lesions and the other concurrent tissue-changes can stand to each other in the relation of cause and effect, as has been suggested; or how morbid conditions, regularly associated with the bone lesion and in proportion to it, can be regarded as accidental. The explanation of the earlier and greater prominence of the bone changes lies in this, that the bones, being in the most active stage of their growth, exhibit the departure from the normal most clearly at a time when the coexisting changes in other organs and tissues are smaller and less visible.

Many are the causes to which the production of rickets has been attributed. Faults of diet, setting up gastro-intestinal catarrh, impaired digestion, vomiting and diarrhoea; want of light and fresh air; deficient clothing, dirt, and general bad hygiene; syphilis; inherited tendency; have all been credited with more or less reason as concerned in the genesis of the disease. It appears certain that, as a rule, several factors are engaged, and amongst them many of those enumerated above. These factors, however, are not all in action in every instance, and they are not, therefore, all essential, nor equal in constancy and potency.

Climate, Season, and Locality.—Although rickets seems to exist in all parts of the world, its occurrence is clearly influenced by climate, and chiefly in respect of dryness, sunlight, and warmth. Thus, it is most common in the temperate zone, especially in cold, damp, cloudy regions such as England, and certain portions of Germany, Italy, France, and North America. It diminishes in frequency as high northern latitudes are approached, and, on the other hand, declines towards the south until it almost disappears in the tropics. In Australia, rickets was believed practically not to exist; but in 1891 it was shewn by Mr. Muskett to be prevalent in the large towns, and cases have been met with even in the bush. The disease appears to be affected by season; cases are more frequent and more severe during the dark, cold winter months when the children live largely indoors, and lack light, fresh air, and warmth; that is, when vitality is lowest. With regard to locality the chief point made out is, that the disease is, generally speaking, one of great cities—of London, Manchester, Liverpool, Glasgow, Vienna, New York; and that in this country it is especially prevalent in the towns of the great manufacturing districts of Lancashire and Yorkshire, the Black Country, Scotland, and Wales; in these regions of smoke and darkness the

mothers are largely workers engaged away from home, and their children are chiefly brought up by hand.

Influence of Sex.—Statistics as to the relative frequency of rickets in boys and girls vary; but the general outcome of them seems to be that sex exerts no influence, and that the disease is distributed evenly between the two sexes.

Inherited Tendency.—No satisfactory evidence has been produced to shew that rickets is ever transmitted from the parents to the children. Rickets dies out with childhood, and it is hardly likely to be handed down to the offspring of mature persons. The influence of heredity is probably limited to the transmission of a weakly constitution, or to some factor of imperfect nutrition of the foetus in the womb. That heredity is not an essential or constant factor is shewn by the observation that the children of perfectly healthy persons become rickety; in the vast majority of cases, indeed, rickety children are born of parents who do not exhibit the smallest trace of past rickets. Seigert, however, believes heredity to be an important etiological factor, and maintains that it is transmitted chiefly by the mother. This writer states that the severest types of rickets is met with in breast-fed children, who have a strong hereditary predisposition.

Congenital Syphilis.—It is now very generally agreed that rickets is not a simple expression of congenital syphilis, as Parrot contended; the evidence to the contrary is conclusive. In the majority of cases these children bear about them none of the well-established signs of congenital syphilis: the eruption, the snuffles, the linear scars, the pegged teeth, the keratitis are wanting. In many cases, moreover, the history seems absolutely beyond suspicion; and, conversely, many children who suffer from congenital syphilis are not rickety. It is clear, then, that syphilis is not a constant, invariable, essential factor. Congenital syphilis modifies rickets, it does not create it. The cases in which it does play a part have special features of their own. The child is puny and wasted, and it presents some of the distinctive signs of syphilis; and to these cases especially belong the craniotabes of Elsässer and the boss-like projections of the frontal and occipital bones in their most extreme form; although they are not absolutely limited to the syphilitic variety. Possibly the enlargement of lymphatic glands, liver, and spleen, met with in some cases, may prove to be rather a syphilitic than a rhachitic change.

Rickets an Infective Process.—Mircoli found staphylococci and streptococci constantly present in the mouths of infants and the mammary ducts of nursing women. He believes that if the alimentary canal of the infant be disordered these micrococci, usually innocuous, become pathogenetic and may produce rickets. They then enter the system and attack the parts of greatest functional activity, among these the nervous system and the epiphyseal ends of the bones. The osseous lesions in rickets he looks upon as the result of a chronic osteo-myelitis; by injection of small quantities of staphylococcus cultures into rabbits of a week old he has produced hypertrophy of the epiphyses.

Bad Hygiene.—Defective hygienic conditions are largely concerned in the production of rickets. The great incidence of the disease upon the population of large cities and amongst the poorer classes there, and the comparative rarity of rickets in the bright, sunny climates of the south, where life is spent largely out of doors in fresh air and sunshine, afford sufficient evidence of this. The want of sunlight and warmth appears to tell especially upon children of southern race when reared in cold and uncongenial climates. The children of Neapolitan parents, for example, brought up in the great cities of America, are stated by Snow of Buffalo to suffer from rickets to such an extent that even those brought up at the breast do not escape. Foul air, want of light and sunshine, defective cleanliness, and lowered bodily warmth from scanty clothing favour the production of rickets by degrading nutrition.

Although, however, these influences of defective hygiene are frequently concerned in the production of rickets they are not constantly present, and are not therefore invariable or essential factors. Many cases of rickets arise in patients who live under excellent sanitary conditions, so far as air and light and cleanliness and warmth are concerned; a child may enjoy these in perfection and yet become rickety: and again a child may not become rickety, although brought up under the most unhealthy external conditions. These external conditions of defective hygiene must therefore be regarded as influential, but not essential; in extreme cases, however, they are generally at work.

Disorders of digestion appear to play a part in a number of cases; at any rate symptoms of gastro-intestinal disorder—flatulence, vomiting, diarrhoea, and offensive stools—not infrequently precede the distinctive signs of rhachitic change. They are not, however, constant accompaniments; numbers of children become rickety who have no such antecedent gastro-intestinal disturbance, and many who do suffer from it do not become rickety. Further, when the disturbance is extreme, and vomiting and diarrhoea are severe and prolonged, the result is not rickets, but general atrophy; and examination after death in fatal cases shews little or no evidence of the characteristic changes in the bones. It appears, then, that digestive disturbance of this kind is only effective when it is not extremely prolonged and excessive, and when it coincides with particular faults of diet. It probably acts by removal of certain special elements which are the least quickly and rapidly digested and absorbed. Digestive disturbance is not, therefore, an invariable and essential factor in the production of rickets.

Faults of Diet.—The vast majority of cases of rickets arise in connexion with errors of feeding. The fault of diet is not only the most common and potent cause, but sometimes it is the only cause. Rickets is produced as certainly by rhachitic diet as is scurvy by a scorbutic diet. This is seen in those by no means uncommon cases of children born healthy and of healthy, well-to-do parents, and brought up under perfect hygienic conditions so far as air, light, cleanliness, and warmth are concerned, who yet become rickety when brought up on artificial food. The

only fault we can discover is the dietetic fault; and such cases are cured by a correction of the diet, without any other change of hygienic conditions; they are cured, in fact, by antirhachitic diet as certainly as scurvy is cured by antiscorbutic diet.

The effect of diet was shewn in the most striking manner by Mr. Bland-Sutton in the case of rickety animals at the Zoological Gardens; these animals got rapidly well on a change of one condition only, namely, of food. Directly or indirectly, food is probably an invariable factor. The fault, moreover, is one of quality rather than of quantity. A child may be reduced by starvation to the last stage of atrophy, and yet not be rickety; and, conversely, it may be over-fed, fat and gross, and yet extremely rickety. There is a special fault of diet, one which produces a special defect of nutrition, and not necessarily general malnutrition. In this respect it is first to be noted that, in this country at any rate, rickets is practically unknown amongst sucklings. The only instance, within our experience, of rickets arising in a child while at the breast during the first ten months of life was one in which the mother became pregnant during lactation; the suckled infant became rickety, the foetus unborn escaped; rickets, then, may arise if the mother's milk be insufficient, or otherwise defective; all such cases are undoubtedly rare in this country. Holt, however, remarks that the negro children brought up at the breast are many of them rhachitic. Even with congenital syphilis at work the child at the breast does not become rickety. If breast-fed children become rickety it is after weaning; or it may be that undue prolongation of lactation and the resulting loss in the nutritive value of the milk may be responsible. The disease, however, occurs almost entirely amongst children brought up by hand.

The exact nature of the diet fault which lies at the root of the rickety condition has been the subject of many hypotheses and much controversy. Certain broad facts, however, have been established with regard to it, which throw great light upon the matter. In the first place, children fed almost entirely upon farinaceous preparations—oatmeal, corn-flour, bread, patent foods, with little or no milk, even if such diet produce no digestive disturbance—certainly became rickety. Similarly in the case of animals, Mr. Bland-Sutton observed that the young monkeys at the Zoological Gardens in London, if deprived of their mother's milk and fed entirely upon vegetable food, chiefly fruits, become rickety. Two young bears fed exclusively upon rice, biscuits, and raw meat, of which latter they hardly ate, died of extreme rickets. It is not a diet limited to vegetable food only which is associated with rickets. The artificial production of rickets in young animals by Guérin, who substituted meat for mother's milk, although impugned by the later experiments of Tripier, has been remarkably confirmed by experience at the Zoological Gardens. For many years the lion whelps have been weaned early, and put upon a diet of raw flesh only; they have invariably become rickety to such an extreme degree that it has been found impossible to rear them. The condition is a true rhachitis; there is the same feebleness of muscle,

the same debility, laxness of sinew and bending of bones; and the identity of the morbid changes has been fully established.

The potency of such diets in the production of rickets has led to many hypothetical explanations of the exact nature of the defect in them; whether this be negative or positive, the want of some necessary ingredient, or the presence of some noxious ingredient which perverts nutrition, especially that of growing bones. The explanation which at first suggested itself was that as the bones are soft and deficient in mineral matter, and especially in lime salts, a want of lime salts in food is the cause of the deficiency in the bones. Chossat and Milne-Edwards produced curvature of the bones in animals by privation of earthy salts, but the characteristic features of rhachitic bone were shewn by Friedleben to be wanting in such cases. Others, however, as Voit and Baginsky, claim to have established the existence of true rickety change by such treatment.

While admitting that rickets may be produced artificially in animals by absolute privation of lime, yet that the want of lime, at any rate in the form of hydrate or carbonate, is not in itself the essential cause of rickets, as we see it in children, is proved by conclusive evidence. In the first place, rickets is extremely common in the limestone districts where the drinking-water is so heavily charged with lime that the children must necessarily take abundance of it; moreover, numbers of children become rickety who have lime-water regularly added to their food. Secondly, according to Dr. Luff's analysis, the foods upon which children are especially liable to become rickety, such as the farinaceous foods, are rich in lime and also in phosphoric acid; and cow's milk is richer in these than is human milk. So that not only will abundance of lime salts in the food not prevent the development of rickets, but as a matter of fact the disease is usually associated with a full supply of these materials.

The close association of rickets with a farinaceous diet suggested the idea that lactic acid might be the evil agent: starch, imperfectly digested, ferments and lactic acid is formed in excess, which, by uniting with the lime about to be deposited in the bones, is supposed to carry it off in soluble form: or, according to another view of its action put forward by Heitzmann, it irritates the ossifying tissue and stimulates growth when the material necessary to complete the structure is wanting. Lactic acid is said to have been found in the tissues of rickety animals and in the urine; and Heitzmann states that by its administration he has produced the condition directly. This formation of lactic acid, however, has not been confirmed; and the hypothesis is improbable because rickets arises in children in whom there is no apparent disorder of digestion to favour lactic acid fermentation, who digest the starch or maltose thoroughly, and even wax unduly fat thereon. Moreover, according to repeated observations of our own, the rickety state disappears, and health is restored whilst the farinaceous diet is continued unchanged except by the addition to it of certain nutritive elements in which it is deficient. These clinical experi-

ments shew conclusively that the starch cannot of itself be actively harmful. Again, rickets arises in animals fed on a diet—such as lean meat alone—which is not productive of lactic acid; and, lastly, if lactic acid did exist in the blood it would be at once neutralised by alkali there. The fault in diet which is the chief factor in the production of rickets is clearly, then, neither deficiency of lime, nor an excess of starch, nor lactic acid generated from it.

An examination on the one hand of the foods on which children grow rickety, and on the other of the additions to diet by which the condition is cured, throws light upon this point. Certain defects appear to be constant. An analysis of the foods on which rickets is most frequently and certainly produced—such as the various farinaceous foods, domestic or patent, with a small amount of milk, skim milk, condensed milk, artificial foods with desiccated milk, and the like—shews invariably deficiency in two of the chief elements so plentiful in the standard food of young animals, namely, animal fat and protein. The only exceptions to this conclusion appear to be the cases in which the foods that do contain a sufficient quantity of these elements—as cow's milk, for example—produce digestive disturbance, vomiting, and diarrhoea, which disturbances lead to the loss of much of the material ingested. In such cases, moreover, the elements most slowly digested, those, that is, most slowly brought into a fit state for absorption—namely, the fat and casein—would be most largely drained away. Not only so, but to substitute a food deficient in these more hardly digested fats and proteins, which, to ease the digestive difficulty, is almost always done, is to reach the same pathological result by a different route. At the Zoological Gardens the food on which young bears and monkeys become rickety—namely, biscuit, rice, and fruits—is markedly deficient in protein, and fat is practically absent. The food of the lion cubs, which became rickety on a diet restricted to raw flesh, was almost destitute of fat, and was poor also in earthy salts, although rich in protein. The meat was that of old horses almost entirely destitute of fat, and once a week lean goat's flesh. The bones were found to be proof even against the teeth of the adult lions, and those of the cubs were powerless against them, so that the cubs got from them neither marrow-fat nor earthy phosphates. In this case the diet was not deficient in protein, but in fat and earthy phosphates. The history of these lion cubs is very significant: with the exception of a single litter, suckled by the dam ten years before, the cubs brought up on horse-flesh in this way invariably died—the cause of death being, as invariably, extreme rickets. More than twenty litters had been lost in this way. The feeding of the last litter of lion cubs was begun in the usual fashion. The dam had very little milk, and at the end of two weeks the cubs were weaned entirely, and were then put on horse-flesh as usual. They quickly became rickety, and the muscular weakness, as well as bony deformity, were extreme. The malady advanced rapidly and one cub died. Then, by the advice of Mr. Bland-Sutton, milk, pounded bones, and cod-liver

oil were added to the raw meat, which was continued exactly as before; they were kept in the same dens with the same amount of warmth and light and air, and, with the single exception of the addition to the diet, no change of any kind was made in the regimen. The change in nutrition which followed was immediate and remarkable; in three months all signs of rickets had disappeared, and the animals grew up strong and healthy—a unique event in the history of the Society. The experiment seems a crucial one, and decisive as to the part played by fat and bone salts, with some casein and lactose, in the production and cure of rickets.

That rickets frequently follows the prolonged vomiting and diarrhoea provoked by cow's milk, is consistent with this estimate of the effect of deficiency of fat and protein in its production. For as the fat must be emulsified or saponified before it can be absorbed, and as the protein in like manner must be converted into peptone, these elements would be drained off rather than the lactose and salts, which are in solution and ready for immediate passage into the circulation. The result would be a great privation of fat and protein.

Of the three elements of food the imperfect supply of which is found to be associated with rickets, fat is probably the most frequently, if not invariably deficient. The abundance of fat in milk, of the whole solids of which it forms one-fourth, points to the extreme importance of it in the nutrition of growing animals. If fat be removed from the milk, as in "skim" milk, rickets follows. The curative power of cod-liver oil is evidence in the same direction; and it is interesting to find in this connexion, as recorded by Remy, that in Japan, where oils of fishes enter largely into food and children are kept partly at the breast up to five years old, rickets appears to be unknown. Animal fat probably serves some special purpose in the nutrition of the growing structures of a young organism. It is found in all cells, it is probably essential to all cell life and growth, and it is further evident that fat formed in the body out of the carbohydrates cannot, for structural purposes, take the place of the animal fat supplied in food. Phosphate of lime, again, is essential to every tissue, and in rickets is found to be deficient in the bones and viscera. Protein, again, is essential to the vitality of protoplasm, and indeed to the activity of all vital processes, and is therefore essential to the proper use of the other elements; but if it be in excess while the other elements are deficient, it may actually intensify the progress of rickets by stimulating the processes of tissue development which, in the absence of other materials, cannot be duly carried out. Therefore a diet deficient in the element of animal fat and, in some cases also, deficient in protein and in earthy salts—one or both—would explain not only the faultiness of bone, but also the feebleness of muscle, the anaemia, the catarrhal tendency of the mucous membranes, and the nervous irritability. All tissues—not those of bone alone—are ill-nourished if the above structural elements are deficient.

The general pathology of rickets may, perhaps, be summed up as

follows:—Rickets is a disease in which all the leading structures of the body suffer both from defective and from perverted nutrition. This is mainly the result of faults of diet, and consists in a deficiency of certain elements in food. It can as certainly, though more slowly, be cured by the addition of such elements to the food as scurvy can be cured by the addition of antiscorbutics. The chief and constant defect appears to be an insufficient supply of animal fat, and therewith also, in certain cases, a deficiency of earthy salts in the form of phosphates; at the same time, if animal protein be deficient the disease is intensified. The development of the disease is also favoured, and it is aggravated in degree by evil external hygienic conditions, such as want of light, warmth, and pure air. The disease is modified in character by the concurrent existence of congenital syphilis or of scurvy. Lastly, in some cases in which the fault of diet is not sufficiently great to cause it unaided and alone, the appearance of the disease is determined by the other factors.

The increased vascularity of the ossifying cartilage, and the excessive proliferation of cells observed in rickety bone, is suggestive of a subacute or chronic inflammatory condition; and this view is supported by the experiment of Kassowitz, who produced a state of bone corresponding to that of rickets by inducing hyperaemia in the limb of a growing animal by means of repeated applications of an Esmarch's bandage. The increased fibrosis and cell-proliferation observed in the liver and spleen in certain cases is consistent with this observation, although it is to be noted that these changes have not been found in all instances or in other tissues undoubtedly affected by the rickety condition. The agent in human rickets has been referred to some irritant derivative of food circulating in the blood. It has been shewn that lactic acid derived from carbohydrate food cannot be regarded as the peccant matter; but it may possibly be something formed from altered materials present in excess, because unused in the disorganised condition of the formative process. The profuse sweatings are suggestive of a like cause; while on the other hand the absence of pyrexia seems opposed to this view, although not conclusive against it. It is possible that the abnormal vascularity and cell-proliferation are the results of the impetus of the formative process left unsatisfied in its normal direction for want of materials for the building of the permanent structure.

Morbid Anatomy.—The bone changes have been aptly described by Sir W. Jenner as exhibiting extensive preparation for ossification and imperfect performance of the process. Our observations agree in the main with the opinion that, as shewn by Kassowitz, the rickety process is not merely a defective ossification, but is accompanied also by hyperplasia.

The Cranium.—The skull well exhibits the three great features of perverted ossification; namely, delay, atrophy, and hyperplasia. The margins of the bones remain membranous; spots of thinning, the craniotabes of Elsässer, are perceptible in the parietal and occipital bones; and small bosses or swellings form symmetrically on the frontal, on the

parietal and occipital, and even on the temporal bone. The atrophic lesions of craniotabes consist of wasting and thinning of the inner table, so that the inner surface of the bone, when the dura mater is removed, presents shallow depressions; some of these, in extreme cases, extend through to the pericranium, while the bone around is thin and elastic. These thinned areas have been variously attributed to pressure of the enlarged brain and to that of the pillow; but they are probably patches of bone which have grown imperfectly. The elevations or bosses are developed chiefly from the outer table of the skull, and consist of red, highly vascular, spongy material which yields to pressure. These, in



FIG. 2.—Rickets. A case of extreme bossing of the skull, the rickety changes probably aggravated by congenital syphilis. Front view. Drawn from life.

A. W., aged 3 years and 6 months. (Hospital for Sick Children, Great Ormond Street, 1881. Dr. Cheadle.)

some instances, become more or less completely absorbed; but in most they become organised, and remain as rounded projections or bosses which are characteristic, or they may spread diffusely into more general thickening of the bone; in either case they largely assist in giving to the rickety skull its special character.

The chief features of this skull are as follows: the forehead is broad, square, and projecting, the eminences on each side being prominent and thickened; the top is flattened so that the head looks square or in some cases oblong, the parietal and occipital protuberances being well marked; a groove runs along the site of the suture between the two halves of the frontal bone, and is continued along the vertex to the anterior fontanelle, or beyond it, to the occiput. In some instances there is a distinct want of symmetry, as if the head had been twisted askew, so that the frontal

region projects on one side and the occipital on the other. The head generally is larger than normal, the face by contrast looking smaller; and the upper jaw is narrowed and elongated. The anterior fontanelle is large, and it remains open longer than usual; instead of being closed at eighteen or twenty months, it may be still more or less patent at two or three years of age; a slight opening may indeed be detected as late as five years. The closure of the sutures, except that between the two portions of the frontal, is delayed also; and there is irregular thickening of the margins of the bones, notably in front of the anterior fontanelle: deep venous grooves, often mistaken for open sutures, are found in the temporal region and elsewhere. The teeth come late; in extreme cases



FIG. 3.—Rickets. A lateral view of the skull in the same case as the preceding. Drawn from life.

none may have appeared at ten or eleven months; they often come through in irregular order; they are fragile and delicate, deficient in enamel, subject to early decay, and fall out before their time.

The Thorax.—In the first place, the shape of the chest is modified by the softness of the rib bones and their yielding to pressure. Of these changes one of the most characteristic is a depression which runs transversely from the lower end of the sternum across the chest on each side to the posterior margin of the axilla, about the line of attachment of the diaphragm. When any obstruction to respiration occurs, and in aggravated cases under ordinary conditions of respiration, the chest is further drawn in along this line with each inspiration. This is a result of the yielding of the ribs in the direction of least support. Another depression runs obliquely down the front of the chest along the line of junction of the ribs with the cartilages; and at the bottom of this depression, or

outside it, are seen the "beads" or enlargements of the growing ends of the rib bones.

The result of this giving way of the shaft bones of the chest-wall under atmospheric pressure, at the points where the ribs are least supported by the rest of the bony framework and the solid viscera, is the



FIG. 4.—Three rickety children in the same family. *Vide* p. 94. A photograph from life by Mr. Higham Cooper.

projection of the sternum forwards. This projection is rounded, not acutely angular as in the true pigeon-breast. The latter deformity results from the respiratory difficulties of whooping-cough, of repeated bronchitis, or of post-nasal adenoid growths, and may arise independently of any rickety softness of bone. When these obstructions to respiration occur in rickety subjects the sternal protrusion becomes more extreme.

The enlargement of the ends of the ribs at the junction with the

costal cartilages—the "beads" which collectively form what is called the "rosary"—is the earliest of all the bone changes; the beads have been recognised at birth, and in some instances even in the foetal skeleton. The beads are most marked in the lower ribs, and examination after death shews that they are more prominent on the inner than on the outer aspect of the bone. A slight degree of beading, however, may not be abnormal. Posterior nodosities which appear to arise from partial fractures are also found near the angles of the ribs in severe cases. In addition to these changes in the thorax proper the clavicles are often thickened and more curved than normal; in some instances they present "green-stick" fractures or imperfect splintering with consequent thickening of the shaft.

The scapula in severe cases is curved, so that the posterior aspect is convex in conformity with the convexity of the back.

The Spine.—The back is rounded, owing to the relaxation of ligaments and the inability of the enfeebled muscles to keep it erect; sometimes there is a slight lateral curvature, and the forward lumbar curve is increased. In the early stage both these curves can be made to disappear by traction; but when the child begins to sit up, permanent deformity may result if the rickety condition persist unrelieved.

The Pelvis.—The flat bones are thickened irregularly as elsewhere: when the body is supported erect on the limbs the pelvic arch, compressed between the weight of the body acting downwards from above through the spine and the upward resistance of the thigh bones, gives way; thus as a general result the pelvis becomes narrowed by the pushing forward of the sacral portion towards the pubes, and is more shallow.

The Long Bones of the Limbs: Bones of the Upper Extremities.—The lower ends of the radius and ulna are thickened, both the epiphysis itself and the junction of this with the shaft; thus is constituted the enlargement of the wrists, which is one of the earliest signs of rickets. Similar but less pronounced changes may be found at the upper end of these bones, and at the upper and lower end of the humerus, being more marked at the latter. The changes observable are at first confined to these; but later, as pressure comes into play, and the child, in squatting or crawling, begins to lean its weight upon its hands, the shafts of the bones of the upper and fore-arm become curved and twisted. The force of gravity appears to be aided and modified by the support and traction of the muscular attachments. The "green-stick" fracture previously spoken of is not uncommon in these bones, and in rare instances complete fractures have been met with. In severe cases thickening of the ends of the metacarpals, metatarsals, and phalanges has occasionally been observed.

The Bones of the Lower Limbs.—In these the earliest and most characteristic change, often indeed the only one to be found in minor examples of the disease, is enlargement of the lower end of the tibia from thickening of the epiphysis and its junction with the shaft. In the more pronounced

cases the upper end of the same bone, and both extremities of the fibula and of the femur, are similarly affected in varying degrees.

When the child begins to crawl or walk about the results of weight and pressure begin to appear, as in the case of the arms; and the long bones become bowed. At first the tibia alone grows a little concave on its inward aspect; this increases if the child be allowed to go on walking and standing, and the femur becomes bowed in like manner. In some of the more extreme cases the tibia undergoes a forward curve just above the ankle, and the femur is arched forward in similar fashion. This is probably the result of pressure on the femur upwards and on the feet

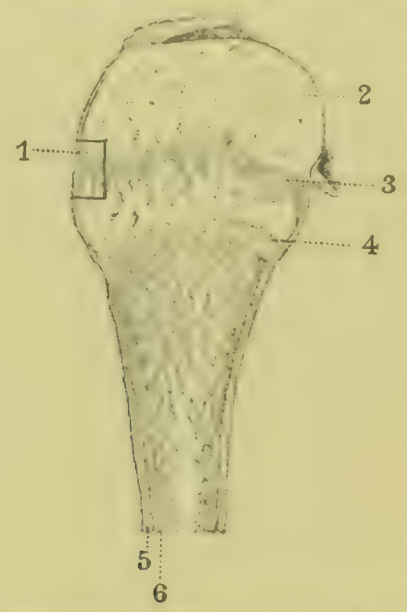


FIG. 5.—Vertical section through the upper half of the humerus of a child aged one year. Natural size. The rectangular area at 1 corresponds to the section, part of which, drawn under the microscope, is shewn in Fig. 6. The cartilage of the upper epiphysis contains enlarged blood-spaces, and at 2 a number of these are closely packed together, shewing the commencement of a centre of ossification. The dark band at 3 represents the epiphyseal cartilage much broader than normal, and provided with irregular processes on its under surface. At 4 is the advancing plane of ossification; this also is broader than in the normal state, in which the layers 3 and 4 are not, as here, separated by a deep layer of soft tissue, but are closely interlocked, and together constitute a plane of tissue of no more than $\frac{1}{10}$th of an inch in depth. The number 5 indicates the periosteum and soft porous bone formed from it after the commencement of the rickets. The number 6 points to the periosteal bone formed before the commencement of the disease. (From an original drawing by Mr. Jackson Clarke.)

backwards as the child is carried in the arms. After a severe attack of rhachitis, a certain degree of coxa vara may result, but this very rarely reaches an extreme degree.

Such are the general features of the bone affection and deformities in rickets. Some of these abnormal conditions disappear with time and growth. In adult life the beads on the ribs are no longer recognisable, the enlargements of the ends of the long bones undergo more or less complete involution, and the deformity of the chest tends gradually to become corrected; but an antero-lateral depression below the nipples persists to adult life in some cases.

The curvatures of the spine and of the long bones slowly straighten,



FIG. 6.—Part of the other half of the same bone as that shewn in Fig. 5, magnified with a low power. (Hartnack, oc. 4, obj. 3.) 1, Fibrous layer of the periosteum; 2, cellular layer of periosteum; 3, bone (imperfectly calcified) formed from the periosteum; 4, cartilage of the upper epiphysis, the disposition of the cells shews that they have multiplied equally in all directions; 5, upper limit of the epiphyseal groups of cells multiplying in a vertical direction; 6, a group of capillary blood-vessels surrounded by calcified cartilage matrix (the matrix of the cartilage is not indicated except where it is calcified); 7, a region similar to that shewn in Fig. 5, but with multiplication of cartilage cells in the calcified tissue which surrounds the blood-vessels; 8, points to the extremity of a blood-vessel where the periosteum dips inwards between the epiphyseal cartilage and the head of the bone; 9, represents part of the epiphyseal cartilage, which constitutes a deep layer in which the cells are irregularly arranged instead of the narrow plane of scalariform groups of cells seen in the normal state. (From an original drawing by Mr. Jackson Clarke.)

and, if moderate, disappear altogether; although in more pronounced cases they remain throughout life. The contraction of the pelvis, however, when extreme, remains; and not infrequently is a serious source of difficulty and danger in parturient women. The bossing of the skull is toned down, although when the hyperostoses are large they remain in a modified form, and are prominent throughout life; the square, projecting forehead and thickened margins of the sutures also continue as permanent indications of the extinct fault of structural growth which gave rise to them. When the rickety condition is severe, growth is defective and the stature short.

In some instances the active changes persist for years. This is well illustrated in Fig. 4, which shews three children of a family aged respectively 10, 7, and 5 years. In each of these the rhachitic signs, which had commenced in the second year of life, persisted in an active state up to the time at which they came under observation. They had all been wofully neglected and ill-fed, and had lived together in a stuffy insanitary room. A younger child aged 14 months, who had come under medical observation from the age of 12 months, and had been brought up in better circumstances, has not as yet developed the disease.

Histological Changes.—The morbid changes in the more intimate structure of the bones consist essentially of modifications of the normal process of ossification; in excessive formation of cartilage and of the proliferating layer of the periosteum, in retardation of the development of bony tissue in these, and in perversion of the process. Bone is formed irregularly instead of by orderly advance of the ossifying column, and it is imperfectly calcified.

With this perversion of the formative process there goes also an absorption of bone already formed. In the long bones these abnormal conditions are exemplified by the greatly increased vascularity of the cartilage—vessels invade the cartilage from the periosteum, and in severe cases enlarge so greatly that they look like haemorrhages or blood-spaces; and also by the excessive development of the proliferating zone of active growing cartilage cells at the epiphyseal end. The cells are increased in number and crowded together; sometimes they actually take the place of the matrix. There is also abnormal shallowness of the columnar zone, where the cartilage cells are being arranged into columns preparatory to their evolution into the medullary spaces of the forming bone. Further, these spaces are themselves irregular in level; and some advance obliquely in the shaft, thus impinging upon and destroying adjacent columns. In extreme cases this latter zone may be quite wanting, and spongy bone approaches the columnar zone of cartilage.

The bone formation takes place not only round the spaces but above and below them also, and small areas of bone may even be found in the unchanged cartilage. The medullary portion of the shaft is more vascular than normal, the interspaces larger.

The formed bone itself undergoes changes; the vessels enlarge and new ones form, which press upon the septa between the medullary spaces

and cause their partial absorption, so that the spaces open into each other. Thus the strength of the bone is impaired, and the slight fractures occur which have already been described. As the septa of the formed bone are eaten away, fresh bone is formed which is deficient in lime. This deficiency is due, not to removal of that material as at first supposed, but, according to the later observations of Kassowitz, to defective deposit: the result is that rickety bone contains only 32 to 52 per cent of lime as compared with the 63 to 65 per cent in normal bone. As the active process subsides, vascularity of the growing portion lessens, and the spongy bone-tissue becomes hardened and condensed by the further formation of new bone rich in lime salts. The junction of the epiphysis with the shaft is sometimes premature and the bone presumably shortened.

So far the description refers to intra-cartilaginous ossification; but changes from the normal occur also in the other form of bone formation, namely, in that from the periosteum. The periosteum strips off the shaft of the long bones more easily, and is more vascular than in health. The outer fibrous layer is thickened, and the inner proliferating layer, in which the bone structure is developed, is the seat of active hyperplasia: the amount of the subperiosteal bone varies from the normal to as many as five or six layers, and it varies likewise in density and in the degree of calcification from spongy, limeless tissue to normal osseous structure. The periosteal formation, which is the sole form which obtains in the flat bones, shews excessive hyperplasia of the proliferating layer and imperfect ossification of like character. This hyperplasia is generally regarded as the result of an inflammatory process, a view which is supported by the fact that with the subsidence of the active rhachitic state more or less induration of bone—sclerosis—follows.

The Ligaments.—These suffer seriously in their nutrition; although no structural changes are found in them, they are obviously enfeebled, and, like the bones, yield under traction and pressure. Thus, the ligamentous structures which bind together the bones of the feet give way as the rickety child begins to bear its weight upon them, and flat-foot results. In like manner the ankles relax under the pressure and the feet splay out sideways; the knee ligaments give way, and knock-knee results; the supporting ligaments of the vertebral column yield, it becomes convex or curves laterally; and thus throughout all joints this laxness and feebleness of the binding ligaments and tendons prevail and alter shape and symmetry.

Muscles.—The muscles are also profoundly affected. This is shewn by their feebleness, which, as has been stated, is in some instances so great that the child may be unable to stand or walk, keep the trunk erect, or in extreme cases to move in bed, or even to raise the head. This lack of muscular tone is frequently illustrated in the parietes of the abdomen, for it repeatedly happens that when a rickety child makes a forced inspiratory effort, the distended intestines pressed down by the diaphragm force apart the two recti and bulge forward between them.

The muscles are seen to be flabby and wasted, and under the microscope the striation is blurred and indistinct and there may be fatty infiltration of the fibres. A complete microscopical and chemical investigation of the tissue-changes in rickets is, however, still wanting.

The Skin.—The skin may exhibit nothing abnormal except some slight pallor; but in severe cases the anaemia is well marked, and in these instances there is also pigmentation. The subcutaneous fat is frequently increased; indeed in the majority of cases of mild rickets the child is as plump or more plump than in health. In a minority of cases, in which there is some general failure of health or a concomitant cachexia such as congenital syphilis, the fat is deficient, the child puny and emaciated. The existence of excessive sweating would seem to indicate the presence of some morbid product in the blood which acts as a stimulant on the glandular structures of the skin; or perhaps the morbid stimulation of gland function may act through the nervous supply, and be a consequence of that hyperexcitability of the reflex actions of the nervous system which is so conspicuous a feature of the disease, and is exemplified by the tendency to laryngismus, tetany, and convulsions.

Mucous Membranes.—These, throughout the whole respiratory and alimentary tracts, are especially prone to catarrh; so that laryngitis, bronchitis, gastritis, and enteritis are common complications. There is marked hyperplasia. The changes in the minute structure of the mucous lining, which are at the root of this abnormal tendency to catarrhal inflammation, have not been made out. All that can be said is that it is due to that vital instability and deficient resisting power which result from imperfect nutrition.

Lungs.—As a result of the falling in of the chest-wall the lungs suffer mechanically. As previously shewn, the depression is brought about partly by the pressure of the atmosphere and partly by the traction of the diaphragm upon the softened bony framework; and this is most effective where the thoracic wall is weakest, namely, at the junction of the ribs with the cartilages, and where the wall is least supported by the viscera. Tracts of collapsed lung are found beneath the line of the grooves of depression, especially under the beads; and with this a compensating emphysema is developed over the anterior borders, where the forward projection of the sternum removes support and promotes distension of the air-vesicles. In addition to this collapse from direct pressure, there may be more extensive collapse from obstruction by mucus when bronchitis has occurred. When cough is violent, as in whooping-cough, the collapse may be so great as to endanger life; and not infrequently, indeed, in that affection it is the complication which determines a fatal issue. The general catarrh of the mucous lining of the bronchi and respiratory tract in many cases has been already mentioned, but the exact changes of tissue which they betray have still to be determined.

The Heart.—In the case of the heart, also, whether in rickets any definite histological changes take place in the muscle has yet to be ascertained; but in view of the great feebleness of circulation which attends

the rickety state there can be no doubt that this organ shares in the general malnutrition. The position of the heart in the chest is somewhat altered ; by the falling in of the thoracic wall the apex is pushed a little outwards towards the left, where, as it comes into close contact with the bead on the rib, a white patch of thickened pericardium is formed by attrition. This is on the left ventricle, not on the right, as is the friction patch of the soldier.

The Liver.—In the majority of cases the liver is of normal size, although its margin may be low in the abdomen, owing to depression of the diaphragm by the contraction of the thorax. In certain instances, which, however, in our experience are comparatively rare exceptions, the organ is distinctly enlarged and harder than normal. This is due to a diffuse fibrosis, with slight increase of cellular elements and a deficiency in earthy salts, as in the bones. The hyperplasia is probably due to the chronic hyperaemia set up by obstructed circulation through the lungs, the result of contraction of the thorax combined with feeble cardiac power, collapse, and emphysema, which retard the outflow from the portal system. Such hyperplasia is readily set up in childhood, a period when the formative process is active. We have seen such fibrosis in marked degree in a child as the result of chronic bronchitis and emphysema. Or the hyperplasia may be irritative in origin, due to the circulation in the blood of some peccant material, such as has been held to be the active cause of hyperplasia in the bones and of the hyperactivity of the sweat-glands. In certain instances the hyperplasia is of syphilitic origin, especially when accompanied by great enlargement of the spleen.

The Spleen and Lymphatic Glands.—With the enlargement of the liver there is commonly also enlargement of the spleen, due to a corresponding fibrosis with cellular hyperplasia and decrease of earthy salts. As in the case of the liver, the hyperplasia depends either upon the mechanical hyperaemia due to obstructed pulmonary circulation and feeble cardiac power, to irritative stimulation, or to congenital syphilis. In cases in which the enlargement is great, sometimes indeed so excessive that the lower border of the spleen extends below the umbilicus, it is almost certainly due to congenital syphilis. The appearance of such a spleen after death cannot be distinguished from that of the organ in so-called splenic cachexia or anaemia pseudo-leukaemica infantum, the origin of which is sometimes syphilitic, sometimes perhaps malarial, in other cases is referred to as the result of auto-intoxication from the intestines, due to chronic digestive disturbances, and lastly is held by some to be a specific disease. The lymphatic glands in these cases are likewise increased in size, indurated, and fibrous from like interstitial and cellular change.

The Brain and Spinal Cord.—The increased size of the head in rickets is suggestive of a brain larger than normal ; but this increase of size is due chiefly to thickening of the cranial bones. There is no evidence of increased cranial capacity except in cases of separation of sutures when hydrocephalus is present. The actual condition of the brain, even as

to size, is a matter of uncertainty ; and of the histological changes we know little.

There can be no doubt that the nervous structures share in the general malnutrition ; but whether this gives rise to increase of bulk, as in the growing ends of bones, or to diminution, as in the muscles, is not clear. According to some observers the brain is smaller than normal, and the extra space in the cranium is filled by effusion of fluid into the ventricles ; according to others there is enlargement, due to increase of the neuroglia —a fibrosis such as that found in other organs. Dr. Shuttleworth has recently directed attention to the frequency of mental hebetude and backwardness in the rhachitic. These abnormalities, he points out, occur in the children of the well-to-do, and are not confined to the poor.

The Blood.—The blood in rickets, unless there be some complication, shews simple anaemia. The red corpuscles are normal or slightly diminished, the haemoglobin falls to 75 per cent or even lower, nucleated red blood-corpuscles are generally present, and the leucocytes shew a slight increase in number. The anaemia as a rule is proportionate to the other changes characteristic of the disease. When, however, it is extreme, it is associated with enlargement of the spleen, and is often out of all proportion to other changes. This extreme anaemia, coinciding with splenic enlargement, is probably the result of the special cachexia of congenital syphilis ; although it must be allowed that such a connexion cannot be traced in all cases.

The Urine.—The analysis of the urine has yielded very diverse results. The great excess of lime salts stated to exist in it by early investigators has not been found by the later, and it would appear that there is really no important difference between the urine of rickety and healthy children in this respect ; nor can the presence of that lactic acid be detected which has been anticipated in accordance with an hypothesis of the pathology of the disease which is no longer tenable.

Symptoms.—The appearance of a child suffering from well-marked rickets in its ordinary form is distinctive. The square forehead with rounded, boss-like projections on the frontal bone ; the enlarged ends of the long bones—the deformities produced by the giving way of soft and yielding bone, such as the contracted chest, the deformed ribs, the curved spine, the protuberant abdomen, the bent arms and clavicles, the bowed legs or knock-knees, the yielding ankles, the defective, decaying teeth, the stunted growth—are features which cannot be mistaken. In many instances the child is plump or even fatter than normal, others again are thin, wasted, and puny.

In addition to these peculiarities of outward form, the rickety infant is pallid, flabby, and feeble. Muscular debility is indeed one of the most constant and characteristic features of the disease, although little stress has been laid upon it. In severe cases the child, at two years of age or later, is unable to stand or walk, or even to sit up ; if it has been able to walk it has lost the power. Such cases are not infrequently mistaken for paraplegia. Sir W. Jenner relates an instance in which the patient,

a girl of six, could not change her position in bed or lift her arm an inch without assistance.

The feebleness of muscle and softness of bone interfere with the action of respiration, and the lower portion of the chest falls in laterally with each contraction of the diaphragm; the feeble intercostals are unable to lift the soft ribs, which give way under the pull of the phrenic contraction aided by the external atmospheric pressure. This inefficiency of



FIG. 7.—Rickets.

C. G., aet. 2 years 6 months, shewing general deformity and distortion of the bony skeleton, muscular wasting, and debility. (Hospital for Sick Children, Great Ormond Street.) Photographed from life.

the respiratory mechanism greatly increases the danger of the pulmonary diseases to which rickety subjects are extremely prone. The affection of the bones in severe cases is sometimes accompanied by tenderness so marked that the child cries when handled, a sign possibly of the commencing scurvy with which rickets is occasionally associated. Another prominent symptom of rickets is profuse sweating, especially of the head and during sleep; this is evidently accompanied by a sense of heat, for the child throws off its bed-clothes at night and lies uncovered regardless of the cold. There is, however, not only no pyrexia, but the temperature is, as a rule, subnormal; unless there be concurrent scurvy or some inflammatory complication, such as bronchopneumonia. The abdomen is prominent partly from the depression of the viscera, partly from the gaseous distension of the intestines and the feeble condition of the abdominal wall. Moreover, the bowels are liable to be relaxed, the stools

being loose and offensive; sometimes, in the absence of biliary colouring matter, almost white; sometimes green, sometimes of darker colour, and slimy. There is a tendency to bronchial and laryngeal catarrh, so that bronchitis and laryngitis are frequent complications. Nervous symptoms form a marked feature of the disease. Sir William Jenner first noted the exceptional tendency to convulsions, and the close dependence of the convulsions of infancy upon rickets was subsequently established by Dr. Gee. Reflex excitability is exaggerated, and probably the cerebral control of the imperfectly developed higher centres is also defective, so that various forms of spasm are readily set up by slight causes of irritation. Laryngismus stridulus, tetany, and general convulsions stand in special relation to the rickety state.

Laryngismus stridulus is rarely met with apart from rickets, of which,



FIG. 8.—Rickets.

From the same subject as Fig. 7, shewing beading of the ribs, transverse groove, and lateral depression of thorax. (Hospital for Sick Children, Great Ormond Street.) Photographed from life

although not universally present, it is a common symptom. In this condition the reflex apparatus of the glottis is so hypersensitive that spasm is excited by slight stimuli, not only by crying or laughter, but even by a breath of cold air, sudden movement, or the emotional disturbance produced by fright, anger, or the vexation of crossed purpose, such as the refusal or removal of a toy. Attacks are especially apt to occur on the child first waking from sleep, particularly in the early morning. The laryngeal spasm is marked by a sudden arrest of respiration, which lasts usually a few seconds only, and as suddenly ends with a prolonged crowing inspiration as air is again drawn through the narrowed glottis. The spasm varies in intensity; sometimes there is merely a slight crowing sound with each inspiratory effort for a brief space; or the muscles of respiration may remain fixed until the child is gravely cyanosed; occasionally death from actual asphyxia occurs before the spasm is relaxed. An attack of spasm of the glottis is not infrequently

the precursor of a general convulsion, and must always be regarded as a significant indication that the condition of the nervous system has become unstable, and is one in which convulsions would readily be set up. Kirchgasser found in a series of 443 cases that rickets was present in 89 per cent.

Tetany is a curious state of painful muscular contraction, a tonic spasm, chiefly of the hands and feet; it is also closely and especially associated with rickets. In Kirchgasser's series of 283 cases rickets was present in 79·8 per cent. In these cases laryngismus is a constant accompaniment, and tetany often follows an attack of diarrhoea, to which rickety subjects are unusually prone.

It is true that tetany occasionally arises in adult life as a sequel of exhausting disease, as in women during lactation, and as a sequel to diarrhoea or enteric fever. Such cases are, however, comparatively rare, and tetany is especially an affection of early childhood and of the rickety state. In tetany the thumbs are first affected, being adducted and drawn into the hollow of the palm, with tips pressing forcibly against the middle phalanx of the third finger, and even driven into the skin. The fingers are drawn together and overlap, and the palm is hollowed, so that the hand becomes cone-like, "the accoucheur's" hand of Trousseau. The wrist is slightly flexed, and in severe cases, when the muscular spasm is great, the back of the hand and wrist may become purplish, swollen, and even slightly oedematous, from the pressure of the contracting muscles on the venous circulation.

The feet are affected in like manner, but usually in less degree. The toes are adducted, flexed, and overlapping, and the forepart of the sole of the foot is hollowed and concave by the drawing inwards of its borders; the dorsum is arched, and in severe cases swollen, congested, and shiny from pressure, like the back of the hands.

In some cases the spasm extends to the muscles of the trunk, causing slight opisthotonos, and occasionally to those of the jaws, causing trismus. The muscular cramps are painful, and when extreme, acutely so; this we learn from the statement of adults, and it is noted, accordingly, that a child with severe tetany cries incessantly, and screams when handled. The tetanoid state persists during sleep, and even under chloroform. It continues a considerable time, often for weeks, and is apt to recur. Remissions occur from time to time. These, however, are not complete; some degree of rigidity still remains.

The spasm can be excited afresh by pressure on the main artery or nerve—probably on the latter—for the motor nerves are in a state of hyperexcitability, not in the limbs only, but in other parts also. Thus, as first pointed out by Sir T. Barlow, the sharp drawing of the forefinger over the skin in front of the ear, where the facial nerve emerges, in a child suffering from tetany is followed by contraction of the facial muscles. Percussion of a muscle causes a like contraction in it. The electrical excitability of the nerves of the most affected parts is also increased both to faradism and galvanism; and, as Erb has shewn, the mode of reaction

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to the voltaic current is reversed, contraction being first excited by positive instead of by negative closure, and a prolonged "tetanus" contraction following both anodal and cathodal closure—the only condition in which anodal "tetanus" has been observed in man. It is said this increased neuro-muscular irritability may sometimes be found in rickety children who are free from attacks of tonic spasm.

Spasmus Nutans.—Dr. J. Thomson found that rickets was present in 33 out of 35 cases of spasmus nutans. This affection is characterised by rhythmical movements of the head, often, but not invariably, associated with nystagmus. It is not probable that rickets is the exciting cause, but it would appear to be a powerful factor in its production. Parents repeatedly complain that rhachitic children knock their heads, and continually roll them from side to side on the pillows. These symptoms, though primarily due in most instances to uneasy sensations in the cranial bones, middle ear, or to adenoids, are encouraged by the nervous excitability that is associated with the rickets.

Such are the symptoms and physical characters of rickets in its full and complete expression. All these features are not, of course, present in every instance. The disease is met with in every degree, from the smallest indications to the extreme forms.

Slight cases of rickets are liable to be overlooked. A little bending of the ribs, a prominence of the frontal bone, small areas of thinned elastic bone on the occipital or parietal, or soft edges of these flat bones, may be the only manifestations of rickets as far as the bony skeleton is concerned. Indeed, the rib-beads constituting the earliest signs met with in the foetal state may be found alone in the early stage. The concurrence of head-sweats, or laryngismus, or convulsion, or a tendency to catarrh, will be sufficient to establish the existence of rhachitis. In a more pronounced case there may further be some enlargement of the wrists, the girdle contraction of the chest, the bending of the long bones of the limbs. In the most complete and severe form the distortion of the skeleton may be extreme, and the whole series of other symptoms affecting the muscles, mucous membranes, and nervous system which have been described may perhaps be present; but the concurrence of all in the same subject is extremely rare.

Forms and Varieties of Rickets, and Conditions resembling it

Foetal Rickets.—Although the observations of Guérin, Tripier, Lauro, Abbott, and others point to the existence of a foetal rickets, there is still doubt as to its occurrence. Achondroplasia has now been definitely separated from the condition of rhachitis, and recent observations tend to differentiate other cases in which changes, such as softening and partial fractures of the long bones, are found in the womb; but the microscope discloses no proliferating cartilage, no imperfectly calcified spongy bone, no true rickety aberration of structure. These cases have

been traced to congenital syphilis, or to the disease termed osteomalacia infantum or osteogenesis imperfecta. Those, however, who maintain that rickets does occur in intra-uterine life, describe cases in which the long bones are bent and fractures are found. There is also incipient beading of the ribs, which on microscopic examination exhibits all the peculiar characteristics of rickets; the ossification of the cranial bones is also delayed. Escher, on the other hand, after an investigation of 105 new-born infants, including twenty-five autopsies, was unable to support the existence of foetal rickets. To us there seems no intrinsic unlikelihood in the existence of a foetal rickets, and we are inclined to believe that such a condition exists.

Scurvy Rickets—So-called Acute Rickets.—Rickets as a rule progresses slowly and insidiously; in a few cases it begins more abruptly, but the disease never sets in suddenly. In all cases in which this comparatively sudden onset has been alleged, there have been signs of bone changes and muscle weakness of longer standing which had attracted little notice until the process became more active.

There are, however, certain cases which have been called acute rickets from the rapid supervention of such acute symptoms as extreme tenderness of limbs, periosteal swellings, sponginess of gums, haemorrhages. The periosteal swellings are due to subperiosteal haemorrhages, and the condition has been shewn by Sir T. Barlow and one of us (W. B. C.) to be in reality one of scurvy, often, no doubt, superadded to rickets, but in its nature distinct. Infantile scurvy is fully described in Vol. V.

Syphilitic Rickets.—When rickets arises in conjunction with congenital syphilis certain modifications follow. The child is as small, puny, and wasted as when rickets concurs with general starvation; and it will probably have the depressed nose and linear scars associated with congenital syphilis. Further distinctive features are the thinning of the flat bones, or craniotabes; and projections on the frontal and occipital bones, the so-called syphilitic bosses: these appear to be specially well marked in syphilitic cases, and, although met with in other instances in which no history or sign of syphilis exists, it is usually in a minor degree. Sir T. Barlow and Dr. Lees found a certain history of syphilis in 47 per cent; Baxter, in 75 per cent; in some cases there is hypertrophic fibrosis of liver, spleen, and lymphatic glands.

Osteogenesis Imperfecta—Osteopsathyrosis—Mollities ossium.—This condition is characterised by a marked hereditary tendency, and may occur in more than one member of a family. The chief clinical features are, first, a tendency to repeated fractures, which may occur irrespective of any demonstrable injury, are often preceded by much tenderness at the position of subsequent fracture, and accompanied by slight irregular fever. The fractures may be congenital or commence later, and, as in a case under our own observation, first appear about the sixth month. They may rapidly unite, without the formation of any unusual amount of callus, or union may be imperfect and even fail altogether. Secondly, the bones are remarkably soft, and in *x*-ray

photographs appear atrophic and unusually transparent. As a result of these two first symptoms there may be terrible deformity. Thirdly, these cases do not react to the most carefully planned and prolonged anti-rhachitic treatment. In one such case treated steadily for two years fractures recurred until death. Neither do they react to antisyphilitic remedies, and for these reasons we are led to the belief expressed by



FIG. 9.—Skiagram of Osteogenesis Imperfecta. Each femur shews a united fracture.

others that the condition is a peculiar one. Lovett and Nichols have noted the association of the disease with unusually small adrenals, but at present the cause is quite unknown. Some undoubtedly recover, others, completely bedridden, die from intercurrent affections. A case of the kind was described by Dr. (now Sir) Thomas Barlow. (See also p. 75?)

Late Rickets.—Rickets is a disease of the first two years of life, and in the majority of cases appears before the end of the first year; in some instances, however, the disease does not set in until later. In one instance, a boy of ten, the first manifestations were observed at the age of nine.

The signs during life—namely, beading of the ribs, contraction of the chest, enlargement of the ends of the long bones of the limbs—were those



FIG. 10.—Late Rickets.

H. J. L., aged 11 years. (Case under Dr. Cheadle. Hospital for Sick Children, Great Ormond Street, 1880-81.) The disease commenced at 9 years old. The bones shewed typical rickety changes. Post-mortem, *vide Trans. Path. Soc.* 1881, xxxii. 386. Photographed from life.

of ordinary rickets; and examination after death disclosed changes in the bones corresponding to those of genuine rickets. It is to be noted, however, as throwing some doubt upon its intrinsic nature, that the treatment

by diet, so effective in this disease as it occurs in infancy, failed in this instance. Sir W. Jenner recorded instances of the first onset of the disease at a late period, namely, at the ages of three and nine. And recently some remarkable cases were shewn at the Clinical Society of London which had apparently commenced much later than these. The difficulty is to decide whether these cases really belong to the category of true rickets.

Diagnosis.—There is no difficulty in recognising the existence of rickets when it is well marked. The bone-changes and other symptoms previously described are characteristic and unmistakable. In mild cases of the disease, however, when the signs are less pronounced, the condition may be overlooked, and is, as a matter of fact, constantly overlooked; such cases being put down as mere weakness, debility, or anaemia. In these less pronounced cases there may be nothing beyond beading of the ribs, slight enlargement of the wrists, squareness of forehead, too widely open a fontanelle, backwardness in teething, early decay of teeth, head-sweats at night, feebleness of muscle or laxity of joints, and perhaps laryngismus. The occurrence of any one of these signs should excite suspicion, and should lead to a minute examination of other points; a concurrence of several of them would establish the existence of the disease. It must be remembered, however, that rickets may affect one part of the body in pronounced degree, whilst in the other parts the changes may be relatively slight.

With the exception of this mistake of regarding a case of slight rickets as a mere condition of simple debility and anaemia, the most common error, perhaps, is that of mistaking the severe form with great muscular debility for the paraplegic form of infantile paralysis. Rickets, however, is usually distinguished by the fact that although the child may be quite unable to stand, it is able to use its legs freely; and although in those cases in which there is scurvy in addition to the rickets the limbs are kept motionless, the marked tenderness, the dread of movement, the spongy gums, the periosteal swelling or other signs, serve to reveal the true nature of the apparent paralysis. Cretinism is sometimes mistaken for rickets and thus valuable time is lost. The harsh skin, scanty hair, large tongue, the aspect, and spade-like hands should generally serve to make the distinction easy. The form of arthritis described by Dr. Still has been confused with rickets. The swellings of the wrists are mistaken for enlargement of the ends of the bones, and the enlarged lymphatic glands and spleen are explained as rhachitic. Attention to the details of the disease will at once shew that the lesions are arthritic and of an entirely different nature to the osseous changes of rickets. (*Vide* p. 17.)

Prognosis.—With regard to the rhachitic state itself the prognosis is favourable if the patient be placed under suitable conditions. The tendency is towards recovery; the chief question is the degree of bony deformity or stunting of structure which will remain. This will depend partly upon the acuteness and severity of the affection, partly upon the stage at which it comes under treatment. Rickets is not a fatal disease

in itself, yet it is liable to aberrations and complications which endanger life. The child suffering from rickets is in a state of constant insecurity. Not only is there the special tendency to pulmonary catarrh, but the danger of this is greatly aggravated by the soft condition of the bony framework of the thorax, which prevents full inflation, especially under obstructive difficulty ; while the deformity of the chest favours pulmonary collapse. Accordingly all diseases of which bronchitis is a common complication—such as measles or whooping-cough—become far more grave if the child be rickety. Similarly the tendency to catarrh of the intestine adds to the frequency and severity of attacks of diarrhoea ; the instability of the nervous system gives rise to laryngismus stridulus and convulsions which may prove fatal ; while the supervention of scurvy, of grave anaemia, or of anaemia with splenic enlargement adds much to the gravity of the condition and to the uncertainty of the prognosis.

Treatment.—*Prevention.*—Rickets in its ordinary form, being essentially a diet disease, although frequently aggravated or intensified by external injurious influences, is eminently a preventible disease. It cannot, indeed, be stamped out until poverty is stamped out ; rickety diet is cheaper and less troublesome than a non-rickety diet. In great cities especially, where the disease is most rife, milk is dear and often largely deprived of cream, while the other factors of defective health conditions prevail there likewise. The disease can, however, be absolutely prevented in most cases, and its frequent occurrence amongst the children of well-to-do people, although it is less common and less severe in this class than amongst the poor, is a grave reflection upon those responsible for their nurture. Rickets, as shewn above, has its origin almost invariably in certain errors in bringing up by hand—either (i.) in the use of artificial foods which are deficient in the elements most important for structural growth, or (ii.) in the administration of foods which, although they contain the essential elements in sufficient quantity, contain some of them in a form not easily digested. In cow's milk, for example, all the essential elements are present, but the casein is liable to set up gastro-intestinal disorder with vomiting and diarrhoea ; thus the elements in question, although present in ample quantity in the food, are drained away undigested and unused. In the majority of instances the two faults are combined : in order to avoid the recurrence of the disturbance which the latter excited, a diet deficient in the necessary elements is substituted for the sufficiently rich but indigestible one. Thus the morbid state initiated by the first error is accentuated and increased by the means taken to repair it. The great point in the prevention of rickets, especially if the child has to be weaned soon after birth, is to take the utmost care not only to give a food which is properly nutritive, but one which will not by its indigestibility set up gastro-intestinal trouble. It must contain animal fat, protein, and carbohydrates in the proportions which obtain in human milk — that is 3·5, 1·5, 6·34 per cent respectively — in easily digestible form, and salts. The proportion in which these elements exist in any given food

can be easily estimated by consulting the tables of analyses which shew the various constituents. The safest method is to transfer the infant to a good wet-nurse ; but this is an expedient open to the wealthy only, and, even if expense be not a bar, it is not always easy to secure a satisfactory foster-mother. The next best plan is to feed the child on asses' milk, which closely resembles human milk in composition. This should be scalded or raised to the boiling-point as soon as received in order to prevent contamination, and passed through fine muslin to remove the small coagula which occasionally form on heating. Here again, however, the difficulty of expense and supply is often prohibitive. Failing these measures, a mixture of boiled cow's milk and water may be tried, beginning with 1 part of milk to 4 of water, and increasing the strength as the stomach can bear it. A useful device is to add to this milk citrate of soda in the proportion of two grains to each ounce of milk. This renders the clot of casein looser and more digestible. The deficiency in fat and sugar in this diluted milk is brought up to the standard by the addition of cream and sugar of milk. If boiled cow's milk, simply diluted thus, and, if necessary, citrated, produce any gastric disturbance, pancreatised milk diluted with an equal quantity of water should be substituted. After a time the degree of predigestion should be very gradually lessened, by reducing both the pancreatising agent and the length of time allowed for the process, until eventually it is discontinued altogether. This end should be attained in the course of a month or six weeks if possible. If the pancreatised milk be too long continued the function of the stomach becomes seriously enfeebled, nutrition is impaired, and a store of digestive troubles laid up for the future. The same caution holds good with regard to all the predigested foods, patent or other, as well as to peptonised milk ; their continued use is eventually injurious.

The proportion of milk to water may be increased until, by the age of six months, that of two to one is reached. If the child be not able to digest cow's milk, except when so diluted that it cannot be taken in quantity sufficient to supply the necessary nourishment, and if asses' milk or a wet-nurse be not available, raw meat juice and cream may be added to it or substituted for it. In cases in which a moderate amount of curd can be digested, the so-called humanised milk, made by removing a portion of the curd by rennet, may be used. It should be freshly made, and, at first, diluted with one-third water. It ought not to be continued beyond the age of three months, since after that age a larger amount of protein is required, and the child is liable to fail in nutrition ; cow's milk and water should then be gradually substituted by admixture with it in increasing quantity. It is important to ascertain that the milk contains a full proportion of cream, namely, 3 to 4 per cent.

In the early months of infancy farinaceous food should only be given when it is necessary as a supplement to fresh milk, or its equivalent of fresh animal material. The malted forms are to be preferred since they add soluble carbohydrate, namely, dextrin and maltose, as well as the soluble protein gluten. Further, no artificial food of any kind should be

regarded as sufficient in itself alone; fresh milk should be added, or its equivalent of fresh animal constituents.

At six to eight months, when the child is brought up by hand, a malted farinaceous food may form a regular addition to the milk and water; or the finest entire wheat flour, or fine oatmeal, may be used if they are thoroughly boiled and the coarser particles strained off. This should be gradually changed to boiled bread and milk, which by the time the child is twelve months old should form the staple food. At ten or twelve months the diet may be increased by the addition of a cup of chicken- or beef-tea, with a scalded rusk, for the midday meal; and the nutritive properties of the meat-tea may be further improved by boiling in it some simple vegetables, such as potato and carrot, and straining them out before use. Between twelve and eighteen months a little bread and butter may be given, and the yolk of a lightly boiled egg; the midday meal may be advanced to well-boiled white fish, or the soft pulp scraped off a slice of underdone mutton, with potato thoroughly mashed and rubbed through a sieve and mixed with gravy. As the age of two years is approached—or even earlier—at eighteen months, if the child is robust—a milk pudding or custard pudding or a well-baked apple may be given at dinner in addition; a little red-currant jelly with bread and butter, or a sponge cake, makes a suitable addition to milk at tea-time.

Correct feeding on a diet of this kind—namely, fresh food containing all the essential elements in due proportion in a form which a child can readily digest, so that it produces no gastric disturbance—is the chief point in the prevention of rickets. But in addition to this the other canons of healthy existence must be observed also. Sunshine and light, so far as they may be obtainable in this climate, are powerful agents in aiding vital processes, and are of immense importance together with fresh air, large, well-ventilated sleeping-rooms, and warm clothing to body and limbs. These essentials to full vigour of growth are very imperfectly secured even amongst the higher class of people. Nurseries are frequently overcrowded, and not infrequently, for the sake of convenience, the poorest rooms are assigned for the purpose. Arms, legs, and necks are left bare, and light linen clothing used when warm woollen materials are required.

Curative Treatment.—As in the case of prevention so in the cure of rickets, treatment is essentially hygienic and chiefly dietetic. Drugs play a secondary part. As a rule far too much reliance is placed upon them, and children are drenched with cod-liver oil, iron, "chemical food," or lime-water, often to the disturbance of digestion and the impairment of natural appetite, and consequently of nutrition. Such remedies are useful and have their place; but they are by no means essential or of prime importance in most instances. Drugs are in reality chiefly useful in the treatment of complications.

As explained in speaking of prevention, the position is usually as follows:—The child has originally been placed upon a diet which has

set up gastro-intestinal catarrh from the irritation of imperfectly digested fermenting material, such as the massive curd of cow's milk; this leaves behind it a chronic digestive inability, so that diluted milk, of sufficient strength to supply the required nutriment, sets up flatulence, vomiting, colic, and diarrhoea. In order to obviate this some artificial food has been given in place of it, which is deficient in the nutritive essentials; or a dozen different foods have been tried one after the other in blind experiment, with the result of perpetuating the disorder and increasing malnutrition. In other cases a food of imperfect nutritive quality, deficient in fat and protein, has been given from the first; this may agree perfectly perhaps with the digestion, but be eminently productive of the rickety condition; that is, a rhachitic diet. The great object is so to amend the diet that it shall yield protein and fat in digestible form up to the full standard.

The diet to be adopted must be on the lines previously laid down for the prevention of the disease. It may chance, however, that the child is unable to digest the nourishment proper for its age; as, for example, the common aliment of cow's milk and water of strength sufficient for growth and nutrition. In this case the deficiency of fat must be made up by the addition of cream; and the cream should be continuously and gradually increased from a few drops to a dessert-spoonful in each bottle if required, the quantity being regulated according to the age of the child, its power of digesting it, and the amount required to raise the food to the full standard of 3 to 4 per cent. The protein is best increased by the addition of raw meat juice; this is the most easily assimilated of all protein matter, and has the additional advantage of being the most powerful of all remedies for anaemia; probably by virtue of the iron of the fresh haemoglobin. Raw meat juice; should be prepared by soaking finely minced beef in an equal quantity of cold water for half an hour, and expressing all the juice through fine muslin by twisting it. A teaspoonful of this should be added to the bottle for a young child; but as it does not keep well, and must be freshly made each time, it may be given once a day in the larger quantity of a dessertspoonful up to two tablespoonfuls according to the age and requirements of the individual patient.

In the case of the poor, cream, or even good milk containing a due proportion of cream, is out of reach, and cod-liver oil may have to be given in its place. With older children boiled fat bacon, or the liquid fat of broiled bacon, forms a most digestible and satisfactory substitute. If the child be wasted as well as flabby and bloodless, some form of malted food, preferably one made from entire wheat flour, should be added to the milk. In the majority of cases of rickets, even if the diet be such as to comprise the standard elements in correct proportions, an increase in the amount of assimilable fat and protein in the shape of cream or cod-liver oil and raw meat juice will greatly quicken the rate of recovery, care being taken not to overtax the child's digestive powers.

Finally, in cases in which the limbs are so tender as to give rise to

a suspicion of incipient scurvy, and indeed in all cases with marked feebleness and anaemia, it is well to give some fresh vegetable juices. This is best done for young children in the way previously described by boiling potato and carrots in their meat-teas or broths, and straining them. To children of eighteen months or more these vegetables themselves may be given, if well boiled and thoroughly mashed.

Drugs.—It has been already affirmed that drugs play a secondary part in the treatment of rickets. Cod-liver oil is useful in some cases, especially when good milk and cream are not attainable, or not well borne, but it is to be regarded rather as a food than as a drug; so far as remedial power is concerned it appears to possess no advantage over other animal fats, such as those of cream and bacon. Cod-liver oil is frequently given in too large doses, so that it interferes with digestion, or sets up diarrhoea. It is to be remembered that a rickety child is prone to gastro-intestinal catarrh, and one teaspoonful of oil twice a day in addition to proper diet is sufficient for a child two years old. The oil may be rendered less laxative by the addition of an equal quantity of lime-water, with which it forms an emulsion. If, however, the cod-liver oil cause undue looseness of the bowels, it does harm rather than good and should be discontinued. The intestinal flux interferes with the absorption of nutriment, and drains it away. Iron is useful for the relief of anaemia, and may be given with cod-liver oil in the form of steel wine, or syrup of the phosphate or citrate, although these preparations of iron are less effective than raw meat juice as a means of restoring red blood-corpuscles and their haemoglobin. The earthy phosphates are present in ample quantity in milk, and in the farinaceous preparations made from wheaten flour or oats; and they are probably most easily assimilated when administered in food. In some cases of more extreme disease, or where food is taken in insufficient quantity, it may be well to give lime salts in the form of the syrup of lactophosphate of lime, or of the so-called chemical food. Experience shews that lime-water is quite useless in this respect, and that the salt must be in the form of phosphate.

In some instances the bitter tonics, such as quinine and bark, are useful for the purpose of giving tone and appetite; but it is doubtful whether their influence for good compensates the drawbacks which attend their administration to young infants. Iodide of iron, so frequently given, is, in our experience, of no service, and, indeed, harmful from its depressant and blood-deteriorating properties; when given to children for a long time in frequent doses it has been observed to produce purpura and great debility; indeed iodides in any form are clearly contra-indicated in a disease of degraded nutrition like rickets.

Phosphorus, on the strength of its reputed power in consolidating new formations of bone, was adopted by Kassowitz as a remedy for rickets; and it has been used to a considerable extent by Continental physicians, with results which are regarded by the majority of them as highly favourable. It must be given dissolved in olive oil, and in

extremely small doses, beginning with $\frac{1}{125}$ grain, in order to avoid gastro-intestinal catarrh, since it acts as a local irritant of the mucous membrane. In certain cases of extreme severity, perhaps, this additional agent, although directed solely to the relief of bone defect, may be of service in pushing forward repair; in such cases, however, the drug is constantly prohibited by the inflammatory catarrh of the alimentary canal which almost invariably coexists. Loewenheim has attempted to overcome this difficulty by employing a preparation termed phytin. Phosphorus occurs in vegetables as an anhydrous oxymethylene-diphosphoric acid, and this acid in combination with calcium and magnesium constitute the preparation phytin, which contains 22·8 per cent of phosphorus. In doses of from 4 to 8 grains he has found this preparation of value.

Several observers also claim good results from organotherapy. Mendel, for example, has employed thymus, and Aussel thyroid. Stoeltzner, in particular, has found that suprarenal extract controls the sweating, improves the power of walking, hastens the repair of craniotabes and the appearance of the teeth.

Of the value of these methods of treatment we have had no experience. The results of correct dietetic treatment, aided where possible by other hygienic conditions, are so satisfactory that we have found no further measures necessary.

Massage is of great service in the treatment of rickets. It improves the nutrition of tissue by increasing the flow of blood in the parts, thus aiding the advent of fresh nutritive material; it also hastens the removal of effete products by quickening the circulation. The practice of massage is described in the article devoted to this subject (Vol. I. p. 422).

In cases in which the condition borders on scurvy, and there is tenderness of the limbs from persistent engorgement, massage is not available.

Baths, again, are of service in improving the circulation, aiding nutrition, and giving tone to the nervous system. The best method, after the child reaches the age of six months, is to stand it upright in a warm bath of 90° to 98° F., and to apply a douche of colder water at 60° to 70° freely with a sponge for a few seconds. For children of ten or twelve months salt water may be substituted for fresh water. The advantage of allowing the child to sit or stand in a warm bath while douched with cooler water is the avoidance of chill, usually shewn by cold hands and feet, which is readily produced in rickety children; many weakly infants turn blue in a bath of 70° or 80° F., and then the bath does far more harm than good. In all cases the bath should be followed by steady friction before the fire, until surface warmth and circulation are fully restored.

Clothing.—As the temperature of the body in rickets, when no complications are present, is persistently subnormal, the preservation of bodily heat by appropriate clothing is an important element in the treatment. Not only is the temperature below normal, but the cardiac muscle is feeble, arterial tone impaired, and the general circulation depressed. Thus the child suffers from coldness of the extremities, and is easily

affected by surface chill; the cooling process is further favoured by the evaporation of the profuse perspiration, whereby congestion of internal organs is readily produced. The condition of the growing bones is also unfavourably influenced by coldness of the limbs. The underclothing should be of soft woollen material, the arms and neck being kept covered even indoors by long sleeves and high-necked dresses, and the legs by drawers or leggings. The only modification on going out of doors, or for change of season, should be in the matter of the thickness, not in the nature of the material. A night-dress of fine flannel is advisable, so that lighter bed-clothes may be used, and the chilling avoided which is apt to result when the child throws them off.

Fresh Air.—The value of abundance of fresh air in maintaining the health of children is very generally acknowledged—but practice falls greatly behind principle in this respect. Delicate children, especially those with a tendency to catarrhs, and rickety children, are often kept too closely indoors, and the temperature of the rooms and house in which they live is kept too high. The chief reason why the child "takes cold" in such cases is the change from the warm atmosphere of the room to the colder air outside; the skin and mucous membrane being morbidly hypersensitive. The way to remedy this instability and increase their powers of resistance to the reflex disturbances of cold and damp in this chill climate, is not to confine the child to warm rooms, nor to heat halls and passages with hot-water pipes and stoves, but, in ordinary circumstances, to keep down the temperature of the nursery to the reasonable standard of 60° F., or even a few degrees lower. Delicate children of all kinds, and rickety children amongst them, may be taken out of doors even in somewhat severe weather for very short periods, which may be repeated, always provided that great care be taken to keep up the bodily heat by warm clothing, shelter from the wind, and the administration of a little food before going out. Another point of equal or even greater importance in the treatment of rickets is the ventilation of the rooms, and especially of the sleeping-rooms. As mentioned in treating of prevention, nurseries are frequently greatly overcrowded and badly ventilated. In the case of a rickety child the cubic space allowed should be ample or even excessive—800 to 1000 cubic feet, and the room should be warmed by a good open fire or fires, so as to assist ventilation by a free out-draught, and not by stoves or hot pipes in any form, which injuriously affect the quality of the air. No nursery should be lighted by gas, not merely for the negative reason that it consumes oxygen largely, but because the products of combustion which are given off are positively injurious. Candles and lamps burning pure vegetable oil, which consume less air and give off little except simple carbonic acid and water, are comparatively harmless. The electric light, which consumes no air and gives off no products of combustion, is the best of all. The ventilation of the nurseries by the usual means of window, door, and chimney should be aided by Tobin's tubes. Further, if the child live in a town, or in a place in the country which is damp

and cold, or much shut in by trees, he should be removed to a fresh open situation, with dry soil and bright atmosphere; and near the sea if possible. It is desirable that he should remain away from home until the acuter signs of the malady have entirely disappeared.

Light and Sunshine.—The effect of sunlight is probably as potent or more potent than that of fresh air. Light, and especially full sunlight, is as important to the cure as to the prevention of rickets, and the removal of a child thus suffering to a bright and sunny climate is of great value in hastening its recovery.

The treatment of *deformities* comes largely into the province of the surgeon, and it is not necessary to speak of it here at any length. It may, however, be pointed out, first, that care during the early stage does much to obviate deformity; and secondly, that the deformities tend to get well. If the bending be not great in degree many bent bones gradually become straight. If the attack be severe the horizontal posture should be maintained, and the back and limbs be duly and evenly supported and kept at rest, especially if the parts are tender. Partial fractures and acute bending should be treated on the same principle under skilled surgical direction. The use of splints during the early stage is of little value, if any, for the purpose of straightening curvatures or lax joints; but a long splint protruding beyond the foot is of service in some cases to prevent walking. If there be no tenderness of bone, massage is of great value in improving the nutrition of bone as well as of muscle and tendon, and in lessening the ultimate deformity.

Treatment of Concurrent Disorders. — Gastro-intestinal disorders are common, and are of importance as leading to malnutrition in two ways; namely, by imperfect digestion and absorption, and by the draining away of nutriment by means of vomiting and diarrhoea.

Here again the main point in treatment is a careful regulation of diet. Inability to digest the casein of cow's milk is the most frequent direct cause of gastro-intestinal disorder in young infants. The cow's milk must be stopped at once and human milk, or asses' milk, substituted, if possible; or the milk may be pancreatised for a time and diluted—the pancreatisation being gradually reduced; or cream and water may be given, beginning with a strength of 1 teaspoonful to 3 ounces, and increasing it to 1 teaspoonful to 2 ounces. Freshly expressed raw meat juice may be added to this mixture in the proportion of 1 teaspoonful to 4 ounces; or chicken-tea, mutton-tea, or beef-tea given independently as a supplement. If the child is eight to twelve months old, some farinaceous preparation, such as one of the malted foods or bread previously well scalded with boiling water, may be added to the milk or its substitute. The dieting should indeed be conducted upon the general plan laid down beforehand with respect to the prevention and cure of the main condition. If the vomiting and diarrhoea are severe, food of the blandest kind, such as thin bread-jelly, weak chicken-tea, dilute peptonised milk, or asses' milk scalded, should be given in small quantities of half an ounce to an ounce at a time, at intervals

of an hour. If collapse be present, brandy, in doses of 5 to 20 drops in a tea- or dessertspoonful of water, should be given every four hours. Valentine's juice in doses of 10 to 20 drops in a tablespoonful of water is often retained when other foods are rejected, and serves to tide over a crisis.

If the stools contain much undigested matter, such as curdy masses, and are slimy and offensive, medicinal treatment may well begin with a small dose of castor oil—diluted with an equal quantity of olive oil in the case of young infants—to clear the noxious matter away. This should be followed by a few doses of grey powder guarded by Dover's powder; a sixth to half a grain of each may be given every four hours to a child six months old. If the bowels are at all loose, so that three or more watery evacuations are passed in twenty-four hours, the great point, next to giving fluid diet in small quantities, is to get the diarrhoea under control at the earliest moment by full doses of subnitrate of bismuth —10 grains in 2 drachms of water may be given every four hours to a child six months old. The small doses of 2 or 3 grains usually prescribed are quite ineffectual; and the soluble preparations of the drug are equally so. The bismuth is best given with spirit of chloroform (℥j. to ℥ij.) and syrup (℥xv. to each dose). If the peristalsis be very active, and the discharge from the bowels copious and frequent, the addition of opium is essential. It should be given in the form of nepenthe or liq. opii sedativus, in doses of a quarter to half a drop for a child six months old. Small injections of starch, with or without a drop of tincture of opium, are useful in lessening the irritability of the rectum. If the colon be specially implicated, as shewn by the passage of mucus and blood, simple injections of warm water are useful to wash out the bowel before the administration of the starch enema; they also aid the effect of the bismuth.

Pulmonary Disorders—Bronchitis, Bronchopneumonia, Collapse.—The treatment of these complications should proceed on the ordinary lines adopted in such conditions. It is well, however, to call attention to, and to emphasise the rule that all depressing remedies—such as antimony, aconite, antipyrin, and the so-called antipyretics generally—are badly borne by rickety children, and should be strictly avoided; also that the cure of the rickety state itself by correct dieting should proceed as far as practicable step by step with that of the immediate pulmonary trouble, in order to prevent the recurrence of such disorders in the future.

Nervous Disorders. Laryngismus, convulsions, and tetany which arise out of the rickety state involve a certain risk to life. In these cases remedial measures must be carried out on three distinct lines concurrently.

i. To remove any source of irritation which may be a cause of reflex disturbance. This is found most frequently in the alimentary canal in the form of undigested food, or of an irritated catarrhal condition of the mucous membrane with consecutive diarrhoea. A dose of castor oil, followed by grey and Dover's powders, or bismuth and opium as previously described, with proper dieting, will usually correct the

disturbance. Another common source of reflex irritation of the cord, sufficient to produce convulsions in a rickety child, is that of teething; a timely use of the gum lancet, now too little as formerly it was too frequently and unreasonably used, will often arrest a threatened attack.

ii. To soothe the abnormal excitability of the central nervous system, and keep it dulled until such time as the source of irritation is removed, and the stability of the centres established. For this purpose chloral has more power than any other drug; its efficiency appears to be increased by combination with the bromide of potassium or ammonium. The use of sedatives has been decried, but they are of signal service in enabling the patient to tide over the period of most active danger. To a child six months old chloral may be given in doses of half a grain to a grain, with 3 or 4 grains of bromide of ammonium, every four hours, until the attacks of laryngismus, the carpo-pedal contractions, or the general convulsions are effectually controlled: the bromide must be diminished or omitted after a time in order to avoid exciting the well-known pustular eruption of the skin; but the chloral may be continued as long as necessary, if care be taken to reduce or stop it if undue drowsiness appear.

The attacks of laryngismus and general convulsions are usually completely arrested after a short time by chloral and bromide, if given in sufficient doses and the patient be brought fully under their influence. If convulsions are so severe that the mixture cannot be taken by the mouth, the same drugs may be given by rectum in larger doses as an enema; namely, 2 to 3 grains of chloral and 4 to 10 of bromide of ammonium (according to age) in half an ounce of water. This measure is even more effectual and more rapid in action than administration by the mouth. The actual attack of laryngismus, which is occasionally fatal, may be arrested by dashing cold water on the face.

iii. The third point in treatment is, as before, to proceed with the cure of the rickety state by the appropriate diet and regimen laid down in speaking of the general treatment of the disease; thus we shall remove the fundamental fault which lies beneath all the various manifestations of it which have been described.

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ACHONDROPLASIA

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Synonyms.—*Foetal Rickets, Foetal Cretinism, Micromelia, Chondrodystrophia Foetalis, Osteogenesis Imperfecta, Congenital Osteosclerosis*

This is a rare and remarkable example of the obscure affections of bone that occur in foetal life. Figure 11 shews the striking feature of the disease, namely, a form of dwarfism, characterised by disproportionate shortness of the extremities, a large head, pug-nose, prominent nates and abdomen, and fingers diverging from one another like the spokes of a wheel.

A considerable number of cases of achondroplasia are on record, and the peculiarities are repeated with such remarkable constancy that, as

several writers have pointed out, we are able to recognise the condition in ancient Egyptian sculpture, and in the works of the old Masters (2), (6).

History.—Our more accurate knowledge of achondroplasia dates from a paper by H. Müller in 1860, who shewed that the condition resulted from disease of the bone-forming cartilage in foetal life; but as early as 1791 Sömmering had described a case of this nature, and Busch recorded another in 1830, under the title of congenital rhachitis. Parrot suggested the name achondroplasia, Kaufmann chondrodystrophia foetalis, Kassowitz micromelia, and Kundrat congenital osteosclerosis.

Pathology.—The etiology is unknown. Busch, Porak, and others (1) have recorded transmission from parent to offspring, and there appears to be some tendency for the disease to occur in families in which sporadic cretinism and dwarfism exist.

There is no evidence that syphilis, alcoholism, inbreeding or debility in the parents have any influence on its incidence. The condition is not confined to man, being met with in such breeds of dogs as dachshunds, and also in some breeds of cattle and sheep. Dr. Seligmann points out that the Dexter-Kerry cow, a breed in which shortness of the limbs is a marked feature, is peculiarly apt to drop cretinous calves, and that this is invariably associated with placental disease. Further observations upon the condition of the placenta in human achondroplasia would therefore be of interest. Hydramnios may occur, abnormal presentations and premature births are frequent, and then the child may either be born dead or survive a few days only.

Virchow and Eberth looked upon achondroplasia as a form of foetal cretinism. The condition is, however, widely different from ordinary sporadic cretinism, and with few exceptions no disease has been found in the thyroid glands of fatal cases. In these rare exceptions a catarrhal condition of the thyroid cells has been observed. Mr. Shattock's suggestion that achondroplasia is a paracretinous condition is the most probable in the light of our present knowledge.

Whatever the causation may be, it would appear that this disease runs its course in early foetal life—probably between the third and sixth months—and that it affects the bones that develop in cartilage during that period. Prof. Symington and Dr. Thomson have pointed out that after the sixth month the bones which are laid down in cartilage do not suffer, and that all the membrane bones escape. It is possible, however, that this only represents a general rule, for there appears to be some evidence that the condition may remain active in some cases until after birth. There is little doubt that some of the foetuses are killed in utero by the disease, if it be in an acute form. On the other hand, when they survive, and reach adult life, the phenomena observed after birth are the outcome of a process which was originally more chronic and has ceased to be active. On account of the early onset, certain bones only are affected, but unfortunately these include some of the most important in the formation of the skeleton. Thus, the long bones of the extremities, the innominate bones, the ribs, the posterior part of the skull and its

base are affected. On the other hand, the sternum, costal cartilages, vault of the cranium, clavicles, scapulae, and vertebral column escape.

Morbid Anatomy.—The pathological changes occur in the process of endochondral ossification. The normal parallel arrangement of the cartilage-cells is disturbed, the growing junctions are not properly vascularised, and in some cases the epiphysis is separated from the diaphysis by an ingrowth of connective tissue from the periosteum. Should this latter process be complete, growth there necessarily comes to an end. Periosteal formation of bone continues, and there is sometimes a very considerable deposit of subperiosteal bone in the neighbourhood of the articulations, and a resulting enlargement of the ends of the shafts of the bones which may, as in the case of the elbow, considerably limit the mobility of the articulation. In Fig. 12 the density of the bones and the undue curvature which is sometimes present are shewn. Kaufmann divides achondroplasia into three different varieties according to the character of the changes in the cartilage:—(1) Chondrodystrophia foetalis hypoplastica; (2) chondrodystrophia foetalis hyperplastica; (3) chondrodystrophia foetalis malica. In the hypoplastic form the epiphyses are not enlarged, in the hyperplastic they are enlarged, and in the third variety there is marked softening of the cartilage. Dr. Ballantyne is disposed to keep each type at present distinct from one another.

Turning now to some of the details in the affection of these various bones, the first for consideration are those of the skull. The basilar process of the occipital bone and two parts of the sphenoid bone undergo premature synostosis, and as a result there is shortening of the base of the skull, and the external auditory meatus passes directly inward rather than forwards and inwards. Kaufmann points out that there is also shortening of the nasal and ethmoid bones. In striking contrast the membrane bones are well developed, and as a result the vault of the skull is of disproportionate size, and the face appears unusually small. The posterior part of the inferior maxillary bone is in some cases shorter than normal, and not uncommonly the lower jaw projects beyond the upper. The ribs are usually beaded from expansion of the ends of their diaphyses. The implication of the pelvic bones leads to a deformity which is of the greatest obstetric importance. The sacral promontory projects unusually into the inlet of the pelvis, and from the shortening of the iliopectineal lines and the slight development of the wings of the innominate bones there is a manifest diminution in the cavities of both false and true pelves. Most important of all is the extreme contraction of the true conjugate at the brim, which seldom exceeds $2\frac{3}{4}$ inches, and may be as small as $1\frac{5}{8}$ of an inch. Another result of the alteration in the pelvic bones is shewn in the peculiar figure of an achondroplastic dwarf. The acetabula, from the shortening of the iliopectineal lines, lie unduly near the sacro-iliac synchondroses, and the femora are thus attached to the skeleton in a plane posterior to that of the normal individual. The nates accordingly are unusually prominent. The sacrum is less vertical, and this, coupled with the prominence of the buttocks, gives the superficial

impression that there is lordosis of the spine. This is not, however, the case, the vertebral column being if anything less curved than normal. The stunting of the long bones is well exemplified in the measurements of the case pictured in the text (Fig. 11). The age of the patient was seven years, and the height 31⅛ inches. The length of the humerus was 2½ inches, the length of the ulna 3⅞ inches. From the anterior superior spine to the adductor tubercle measured 5½ inches. The length of the tibia was 5⅙ inches. From the heel to the tip of the great toe measured 5 inches.



Fig. 11.—Photograph of a characteristic case of Achondroplasia. By Mr. Higham Cooper.

The digits diverge from one another, and are nearly equal in length. The point mentioned by Dr. Thomson, that the divergence is greatest between the middle fingers, and is a result of the enlargement of the end of the bones, is illustrated in Fig. 12. This sign is not pathognomonic.

Clinical Features.—Females are rather more frequently affected than males. The stature is much dwarfed, and it is seldom that an adult reaches 4 feet in height. At birth the disproportionate size of the head may cause much difficulty in parturition. In some cases weakness of the back has been recorded, or the child has been described as generally misshapen.

The general health in infancy may be good, but it is soon recognised that growth is deficient. The cranium appears unusually large, although the measurement is approximately normal. In Drs. Rankin and Mackay's case, for example, the circumference of the skull was 20¾ inches, or normal for the age. The arrested development of the base makes the vault of the skull unduly prominent, and the contrast is rendered more striking by the small pug-shaped nose which projects from beneath an overhanging forehead. In some cases the tongue protrudes from the mouth, the vault of the palate is high, and the lower jaw may be underhung. The expression of the eyes is bright and the intelligence is good, thus contrasting with the condition in cretinism. There is a copious growth of hair. Dentition is normal, and the skin usually soft and natural to the touch, although it may be redundant over the extremities, a point illustrated around the ankles in Fig. 11. In a few cases

there has been swelling and oedema of the subcutaneous tissues. The trunk appears grotesquely long and large, and is, on account of the pelvic deformity, set abnormally upon the lower extremities. A side view shews the results; there is a long straight back ending in prominent buttocks behind, and a protuberant abdomen in front. When vertical the upper extremities reach to about midway between the crest of the ilium and the great trochanter, and end in shortened hands with spoke-like digits. The individual comes to an abrupt termination in the strangely short, thick, and frequently curved lower extremities, which



FIG. 12—Skiagram of the forearm and hand of the same case as in Fig. 11. It illustrates the shortness of the long bones and the "trident hand." By Mr. Higham Cooper.

end in a pair of large, serviceable, rather flat feet. These short extremities bring the centre of the body distinctly above the umbilicus.

The other functions of the body are, as a rule, natural, and although with advancing years the abnormalities are accentuated, all the cases under my observation have been happy in mind, alert, rather sensitive about their stature, and, as might be expected from their unusual relation to their surroundings, quaint in character. They are remarkably active; for their muscular power is good, the bones though short are strong, and their hearts have only a small area to supply with blood. The gait is waddling, and resembles most nearly that in double congenital dislocation of the hip-joint. In some cases other congenital abnormalities have been

observed. Nathan records congenital hernia, and a high arched palate, Kassowitz congenital dislocation of the hip.

I have recently had under my care a girl aged 15 years, 41 inches in height. Her physiognomy and limbs are of the usual achondroplastic type, and at birth her head was noticed to be unusually large. At the age of $2\frac{1}{2}$ years she had two fits, and since then, as her mother assures me, she has not grown properly. In addition to the evidences of achondroplasia during the last two years, a spastic paraplegia has gradually appeared. The reflexes of all four limbs are much increased, and the plantar responses extensor. The left leg is not only spastic, but there is also some contracture. The mental state is good. This case is unusual, for apparently this patient must, according to the mother's statement, have grown to some extent during the first $2\frac{1}{2}$ years of life. The family history was a good one, and both parents were above the ordinary stature.

Diagnosis. — The diagnosis of achondroplasia is seldom difficult except at a very early age. *Rickets* does not produce the remarkable symmetry, the disproportionate shortness of the limbs, the divergence of the fingers, and short hands. In rhachitic dwarfism there are distortions of the bones rather than arrest of development. In achondroplasia dentition is early, and there is not such backwardness in walking as is met with in rickets. The general health is good, whereas the constitutional symptoms of rickets are usually definite. Rickets may undoubtedly occur in association with achondroplasia, and the combination be puzzling, but a careful study of the various deformities will enable a diagnosis to be made. *Sporadic cretinism* has also important distinguishing features. Growth in this condition is much delayed, and the limbs are short, but the disproportion does not approach that of achondroplasia. The lack of intelligence, scanty hair, harsh skin, spade-like hands, pads of fat, thick tongue, and dull vacant look are absent in achondroplasia. The *Mongolian type of mental backwardness* is readily recognised by the oblique palpebral fissures, the semi-idiotic expression, lolling tongue, red cheeks, and round head. Growth and mental development are very backward, but the child is well proportioned. The various forms of *infantilism* may resemble achondroplasia in so far as they lead to dwarfism, but the special features of the achondroplastic dwarf are absent. *Ateleiosis* is distinguished by the childish character of the features, and, although there is dwarfism, by the absence of any undue shortness of the long bones in proportion to the vertebral column. In *cleido-cranial dysostosis* the membrane and not the cartilage bones are apparently the ones affected. The anterior fontanelle may persist until adult life, the transverse diameter of the skull is exaggerated, the face atrophied, and the clavicles almost entirely absent. Dr. Ballantyne, when writing upon the subject of foetal bone diseases, especially with regard to the question of foetal rickets and foetal cretinism, makes the valuable comment that we do not know to what extent these two conditions may be altered in character when they attack the foetus. There seems, however, no doubt that

when a case of achondroplasia survives, it can be clearly and certainly distinguished from all other diseases.

Treatment, so far as is known, appears to be useless. For nothing can restore growing tissues destroyed in foetal life. The only hope lies in the possibility that although much damage has already been done, there is still, in some cases at any rate, some power of growth in the affected bones. With this in view thyroid has been administered, but without success.

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GOUT

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Synonyms.—*Podagra; Arthritis Uratica; Goutte; Gicht.*

The name Gout is derived from the Latin *gutta*, a drop, and is associated with the notion that some morbid matter is dropped into the joints. The disease was well known to the ancients, and its chief features have been adequately described by Greek and Roman physicians, from Hippocrates downwards.

Introductory.—Gout is a constitutional disorder characterised by paroxysmal attacks of inflammation of the joints, associated with the

formation of chalk-like concretions in and about the joints. Since the discovery by Wollaston, in 1797, that these concretions contain urates, and the discovery by Sir A. Garrod, half a century later, that the blood and interstitial fluids of gouty persons are surcharged with the same compounds, the opinions of pathologists have gravitated more and more steadily to the conclusion that abnormality in the destiny and disposal of uric acid is a fundamental element in any scientific conception of the gouty state, and a dominant factor in the genesis of its chief symptoms and physical signs.

Uric acid gives rise to morbid phenomena in two distinct ways; namely, in connexion with gout, and in connexion with urolithiasis. In both these conditions there is an aberration of uric acid; but the error is essentially different in the two cases, both as regards its site and as regards its nature. In gout the error occurs in the true interior of the body, above the limitary membrane of the secreting structures of the kidney—that is to say, in the blood and tissues—and the uric acid is precipitated in a state of combination as a urate. In urolithiasis, on the other hand, the error occurs below the limitary membrane of the kidney, in the excreted urine, and the uric acid is precipitated in the free state, on what is, strictly speaking, the exterior of the economy—that is to say, on the surface of a doubling of the external integument.

Etiology.—The conditions which engender, or tend to engender, the gouty state may be divided into those inherent in the individual, or intrinsic causes, and those which operate from without, or extrinsic causes. The intrinsic factors include age, sex, heredity, bodily conformation, and individual peculiarities.

Age.—Gout is in the main a disease of middle life and advancing years. Typical arthritic attacks usually first shew themselves between thirty-five and fifty. Where there is a strong hereditary tendency the onset is often earlier—and young men and even boys at school may undergo typical joint attacks. On the other hand, the first manifestation of gout may be postponed to old age; in these cases the complaint is usually of a mild character, and appears almost as if it were an incident of senescence. It not infrequently happens that gouty attacks shew themselves in greatest frequency and severity in the fourth and fifth decades of life, and that the subsequent decades are comparatively or entirely free from such attacks.

Sex.—Gout is much more common among men than women. Indeed, characteristic gouty paroxysms are rare in the female sex. The difference, no doubt, is due in great part to the different dietetic habits of the two sexes. It is, moreover, held that the catamenial discharges to a certain extent protect women against gout. After the menopause symptoms of irregular gout are not uncommon, especially in women of plethoric habit.

Heredity.—Gout runs strongly in families. Fully three-fourths of the cases of gout occurring among the easy classes can be traced back distinctly to a gouty ancestry. It is noteworthy that although the women of gouty families may escape overt gout, the transmission of the disease

to offspring is more certain through the female than through the male line. A grandson may inherit gout in full force from a gouty grandfather through a mother who has never herself exhibited any gouty manifestation. It is also beyond doubt that gout newly acquired during life by the action of extrinsic causes may be transmitted to the children. It is significant that in such cases the gouty taint shews itself in the later born and not in the earlier born children. The history of gout supplies some important instances of the possibility, so much debated among biologists, of the hereditary transmission of acquired characters.

Bodily Conformation and Individual Peculiarities.—Persons of large frame and vigorous appetite with a tendency to corpulence may be said to have a natural proclivity to gout; and they easily become overtly gouty if they fall under the operation of the dietetic factors and the sedentary habits which favour the invasion of the complaint.

The extrinsic causes of gout include errors of eating and drinking, idle and sedentary habits of life, lead impregnation, and renal disease.

Food and Drink.—Dietetic habits play a large part in the production of gout. Gout is the nemesis of high living. Of solid food the red meats and game are held to be more provocative of gout than the white meats and fish. Highly nitrogenised articles of food favour an excessive formation of uric acid, and therefore favour the accumulation of urates in the blood. Speaking broadly, articles of food of animal origin are richer in nitrogen than those of vegetable origin. They are also more attractive to the palate, and for that reason tempt to gluttonous feeding. The upper classes—who in this country eat meat two or three times a day—provide the largest contingent to the company of the gouty. On the other hand, agricultural labourers and unskilled workmen, who rarely get meat more than once a day, and often only three or four times a week, enjoy almost complete immunity from the ordinary forms of gout. Meat and highly nitrogenised articles of food lead to an excessive formation of uric acid owing to the presence of xanthine, hypoxanthine, adenine, and guanine. In other words, it is due not to the amount of nitrogen or of protein, but to the presence of these purin bases that these articles of diet are harmful.

Of all articles of diet, however, the most provocative of gout are alcoholic beverages. Sir Alfred Garrod expresses himself very strongly on this point. He writes: "There is no truth in medicine better established than the fact that the use of fermented liquors is the most powerful of all the predisposing causes of gout; nay, so powerful, that it may be a question whether gout would ever have been known to mankind had such beverages not been indulged in."

Alcoholic liquors are, however, not all equally potent in fostering the gouty habit; and the difference between them in this respect has comparatively little reference to their percentage of alcohol. Port, sherry, Madeira, Burgundy, strong ales, porter, and stout are much more powerful

factors in the production of gout than distilled spirits. The prevalence of gout in different districts and countries is found to have a close relation to the kind of alcoholic beverage in common use. In Scotland and Ireland, where whisky is the common drink, gout is not so prevalent as in England, where malt liquors are the popular beverages. In France gout is most common in the Burgundy district; while in the Rhenish provinces of Germany—where various classes of hock are consumed—gout is but little known. Champagnes, especially the sweeter sorts, are undoubtedly gouty wines; cider, on the other hand, has not much power in this direction. It has to be remembered that those who habitually use the richer wines and the heavier kinds of malt liquors are also often large consumers of meat, and that these two factors reinforce one another in the production of gout. Hence certain occupations conduce to gout; and butlers, gentlemen's servants, butchers, innkeepers, brewers' draymen and cellarmen are frequent victims to this complaint. It may further be observed that—apart from quality and quantity—the mode of using alcoholic beverages has much to do with their efficiency as producers of gout. Gout is but rarely found among drunkards, topers, and loafing beer-swillers; nor is it a special concomitant of cirrhosis of the liver, nor of other organic tissue-changes due to alcoholism. Gout is rather an incident of the legitimate dietetic use of alcoholic beverages. Persons who acquire gout as a consequence of their dietetic habits are, as a rule, above reproach in regard to their sobriety; but, at the same time, it cannot be gainsaid that their scale of living is habitually high, and that they are of the number of those who "fare sumptuously every day."

The dietetic habits which dispose to gout have their incidence mitigated on the one hand, or intensified on the other, by the general mode of life. Those who lead an active out-of-door existence can carry off with impunity a scale of feeding which involves those of more sedentary and studious habits in the pains and penalties of gout.

Lead Impregnation and Gout.—It is well known that painters and plumbers are liable to gouty manifestations. The association of gout with plumbism is well marked in the metropolitan area. Among hospital patients in London, Sir A. Garrod found that 33 per cent of those affected with gout shewed distinct evidence of having been poisoned with lead. Sir Dyce Duckworth observed that of 136 cases of unequivocal gout among out-patients of both sexes 25 presented signs of lead impregnation. All these were males, and followed the occupation of plumbers, painters, compositors, or workers in lead-mills. This association is much less frequently observed in Scotland and the North of England than in London. Dr. T. Oliver remarks: "We do not see in the north that intimate relationship between gout and saturnine poisoning. Workmen from the south develop it in the North of England. The natives of the north, though equally exposed, seldom become gouty even when the kidneys are affected." (*Vide* also art. "Lead Poisoning," Vol. II. Pt. I. p. 1058.) Sir Dyce Duckworth cites an analysis made by Frerichs

of 163 cases of lead poisoning in the hospitals of Berlin. Among these there was not a single case of true gout. The aggregate of the evidence collected on this point goes to shew that the association of lead poisoning with gout is scarcely noticeable except in a population among whom gout from other causes is prevalent, and that lead impregnation can rarely promote gout except in persons who have already either a hereditary or an acquired predisposition to the complaint.

Immediate Causes of a Fit of Gout.—The immediate occasion of a gouty outbreak is often undiscoverable. The outbreak seems to arise in most instances as the culminating effect of the predisposing factors, and often occurs unexpectedly in the midst of apparent health. The spring and autumn seasons appear favourable to an outbreak. Not infrequently, however, some particular incident or circumstance can be distinctly indicated as the determining cause of the attack. It may be an unusual access of luxurious living or of indulgence in rich wines; sometimes the attack is traceable to some special worry or anxiety, or a fit of anger, or studious effort. Exposure to cold is sometimes the immediate antecedent of a paroxysm; sometimes an accidental injury to a member or a joint lights up the mischief. Whatever the immediate determining cause of a fit of the gout may be, or appear to be, it is obvious that the incriminated causal factor could not have taken effect had not the sufferer been already gouty and constitutionally disposed and ripe for an attack. The occurrence of a fit of the gout, therefore, not infrequently proves the pre-existence of a long-suspected condition of latent gout.

Connexion with other Diseases.—As gouty persons advance in life they sometimes become glycosuric. This circumstance does not usually appear to aggravate their condition materially. Gouty glycosuria rarely proves severe; it is, as a rule, unaccompanied by thirst and diuresis, and is efficiently controlled by moderate dietetic restrictions. In myelaemic leukaemia the quantity of uric acid produced and voided with the urine is greatly increased; and it might have been expected that persons so affected would exhibit a strong proclivity to gout. This, however, does not appear to be the case. I have only encountered one example in which the two diseases were associated; and in that case the patient had been a martyr to gout for very many years before the spleen became enlarged and the blood leukaemic. Sir Dyce Duckworth observed two instances in which gout coincided with leukaemia; in one case the gout was obviously long antecedent to the splenic enlargement; in the other the sequence was doubtful. I am not aware that any case has been recorded in which gout supervened as a clear consequence and result of myelaemic leukaemia.

Uric acid gravel and calculus are sometimes associated with the gouty diathesis; the connexion between these two conditions is, however, by no means close. The great majority of gouty persons never suffer from urinary gravel; and, conversely, only a small percentage of the subjects of uric acid gravel suffer from gout. The geographical distribution of the two complaints is widely different. In Scotland, where gout is

rare, stone is comparatively common. Mr. Plowright has shewn that there is no correspondence between the prevalence of gout and the prevalence of stone in the several counties of England. Some of the counties which have a high mortality from gout have a low mortality from stone. Norfolk, which was the chief stone district of England, was comparatively free from gout. Uric acid gravel and calculus are common in natives of India, among whom gout is practically unknown.

Morbid Anatomy.—The anatomical changes characteristic of gout depend essentially on the uratic deposits which form in various parts of the body, especially in and about the joints ; and on the inflammatory and degenerative processes which immediately or remotely follow thereupon.

When a gouty joint is dissected after death the articular cartilages are seen to be encrusted with a white mortar-like material, which, on analysis, is found to be composed of sodium biurate. This substance is scattered in specks, streaks, or patches on the cartilages. In some instances the deposit is strictly confined to the articular cartilages ; in others it extends to all the structures of the joint—to the ligaments, fascias, tendons, and the synovial membranes ; and the synovial fluid itself is charged with specks and crystals of the same substance. When the joints are enlarged and deformed the deposit infiltrates the tissues outside and around the articulations — namely, the subcutaneous connective tissue and the substance of the skin—penetrating towards the surface and forming chalk-stones and tophi. The synovia is generally found to be thick and scanty ; but sometimes in the case of the larger joints, such as the knees, there is a considerable effusion of fluid into the cavity of the articulation. Remote secondary changes are often discovered in old gouty joints, erosions of the articulating surfaces and thickenings of the ends of the bones. These latter changes, however, are not peculiar to gouty joints, but are identical in nature and character with those produced by repeated and chronic inflammations of the joints from rheumatism or other cause.

The commonest and indeed often the only seats of uratic deposits found after death are the articular cartilages, and these have been the most carefully studied. On close examination it is perceived that the deposit is not really on the surface of the cartilage, as at first sight it appears to be, but is situated interstitially in its substance. This may be demonstrated by cutting a thin vertical section through the cartilage at the site of the deposit, and examining the section under the microscope. It is then seen that the uratic matter is infiltrated through the matrix of the cartilage beneath the free surface, and that it is composed of felted masses of acicular crystals. The deposit penetrates only to a small depth below the surface, usually to less than one-third or one-half of the entire thickness of the cartilage. It is further seen that the deposit is densest close under the articular surface, and that it grows thinner and thinner towards the deeper parts. The deepest layers near the bone, as well as the bone itself, are entirely free from deposit. Where the deposit is thinnest its crystalline structure is most clearly discerned.

When thin sections of a gouty cartilage are digested for some hours in warm water, the white matter is entirely dissolved out, and the tissue to the naked eye reassumes its normal appearance. But when sections thus treated are examined under the microscope, it is seen that in those parts where the deposit was thick and dense the matrix of the cartilage is profoundly altered, and displays an opaque granular appearance, or may even be eroded; but that in spots where the deposit was thin and sparse the matrix shews no change from its normal aspect. These appearances, which have been verified over and over again, prove that the uratic deposits in gouty joints are thrown down in previously healthy cartilage—that the morbid changes found therein are not primary, but secondary, and are due to the pressure and irritation of the deposited matter. Moreover, the fact that the deposit is densest near the articular surface of the cartilage, and thins away towards the deeper layers, indicates that the uratic matter had its source in the synovial fluid bathing the cartilage, and was precipitated from it.

The causal relation between uratic deposits and gouty inflammation of the joints has been minutely traced by Sir A. Garrod. He made an extended series of observations on the post-mortem appearances of the joints of gouty persons whose previous morbid history had been carefully ascertained; and he found that those joints which had passed through an attack of gouty inflammation at some previous period of life always displayed the accusing uratic deposits at the autopsy; whereas those joints which had not been so attacked were quite free from such deposits. On the ground of these observations Garrod formulated the proposition that "*gouty inflammation is invariably attended with deposition of urate of soda.*" This proposition, thus guardedly worded, is probably universally true; but it does not appear to be universally true that a joint, which at some previous epoch of life has undergone an attack of gouty inflammation, will invariably be found after death to exhibit uratic deposits. Sir Dyce Duckworth records the case of a man under his care with chronic phthisis, who had had two attacks of gout in the right great toe-joint; at the autopsy neither toe-joint contained a speck of uratic deposit. This case, however, does not prove that uratic deposits had not taken place during the gouty attacks; it only proves that such deposits are not always permanent, and that, under certain conditions, they may be removed by re-solution. Nor can it be said that the converse of Garrod's proposition is invariably true, namely, that a joint which exhibits uratic deposits after death must, in every case, have undergone an inflammatory gouty attack during life. F. Levison gives the details of the autopsies of twelve cases of chronic Bright's disease with granular contracting kidneys, in all of which uratic deposits, mostly very slight, were discovered in one or more joints. The previous clinical history of the larger number of these cases was either defective or altogether wanting; but, with regard to four cases in which the previous history was ascertained, no evidence was elicited that any inflammatory attacks in the implicated joints had occurred during life. The absence of inflammatory attacks in these

cases is probably to be explained partly by the scantiness of the deposit, and partly by its slow formation.

The relative frequency with which the several joints are affected with uratic deposits corresponds closely with the relative frequency with which they are subject to gouty inflammation as clinically observed. This broad conclusion is clearly brought out in the records of eighty autopsies of gouty subjects made by Dr. Norman Moore. The most commonly affected joints are the metatarso-phalangeal joints of the great toes; and not infrequently these are the only joints affected: then follow the joints of the insteps, ankles, knees, hands, and wrists. The elbows, shoulders, and hips are more rarely affected. Deposits may be present in nearly all the joints of the lower extremities, and yet be entirely absent from those of the upper extremities. Among the rarest sites of uratic deposits may be mentioned the articulations of the jaws and larynx and the sterno-clavicular joints. The most common sites of *abarticular deposits* are the rim of the ear, and the tendons and aponeuroses in various parts of the body. Such deposits are also seen not infrequently in the skin of the palms and soles, on the eyelids, nose, and other parts of the face; also, more rarely, on the vocal cords, the cranial and spinal dura mater, the pia mater, the sclerotic coat of the eye, the fibrous sheaths of the nerve-trunks, and the aortic valves.

The Kidneys in Gout.—It has been long recognised that there are intimate relations between gout and renal disease. The form of kidney disease which is especially associated with gout is that which is characterised by granular degeneration and atrophy of the organs. Hence this form of renal disease is sometimes spoken of as the "gouty kidney," or "the gouty form of Bright's disease." The relations between articular gout and renal disease are both complicated and inconstant. In many cases the renal affection appears distinctly as a sequence of the arthritic. In other cases signs of renal disease precede the arthritic manifestations; or again the two conditions may arise simultaneously. Sometimes, as pointed out above, the necropsy of subjects affected with granular contracting kidneys discloses the existence of unsuspected uratic deposits in joints in which the clinical history had indicated no antecedent inflammatory arthritic attacks. It may be said, generally, that the coincidence of articular gout with renal disease is more common among the poor and in hospital patients than among the easier classes. In cases of saturnine gout implication of the kidneys is almost if not quite invariable. On the other hand, in private practice, and among well-to-do patients, it is quite common to see articular gout, even of a chronic and inveterate character, run its entire course without any accompanying signs of structural disease of the kidneys.

When gouty kidneys are cut open white deposits of uratic matter are seen on the cut surfaces. In the cortical substance the deposit occurs as specks scattered irregularly through the tissue; but in the pyramidal portion the matter is in streaks running in the direction of the tubuli. In both cases the deposit is situated in the intertubular substance; and,

when examined under the microscope, is seen to consist of acicular crystals exactly resembling those found in gouty joints.

Pathology and Chemistry of Gout.—Gout may be regarded as the expression of a peculiar diathesis, or constitutional bias, which is either inherited from a gouty ancestry, or is acquired during life through the operation of extrinsic causes. The distinctive features of the diathesis are a tendency to (*a*) the accumulation of urates in the blood, and (*b*) to the deposition of urates in the tissues.

These two conditions, to which the names *urataemia* and *uratosis* may be respectively applied, are by no means of identical pathological valency. They differ from each other in several important respects. In urataemia the urates or other compound of uric acid circulate in a state of solution in the blood and lymph—in uratosis the urates are deposited in the solid form in the substance of the tissues. In the former condition the urates can only act injuriously, if they so act at all, after the manner of a chemical poison; in the latter condition the deposited crystals must act more or less as a mechanical irritant. Urataemia, I repeat, is not confined to gout; it occurs also in anaemia, leukaemia, pneumonia, and some other conditions, which cannot by any stretch of definition be included in the term gout. Uratosis, on the other hand, is absolutely confined to the gouty state, and constitutes its pathognomonic mark. Uratosis cannot occur, so far as is known, without coexisting urataemia; but it is certainly true that urataemia may exist for long periods without provoking the occurrence of uratosis. From these differences it may be inferred that the conditions of origin of urataemia and of uratosis are not quite the same; and that in uratosis there are some additional factors in operation beside and beyond those which give rise to urataemia. It is evident, moreover, that there is a closer relation between gout and uratosis than between gout and urataemia; and that the conditions which lead to uratosis have a more immediate bearing on the pathology of gout than the conditions which lead to urataemia.

Chemistry of Uratosis.—It has already been shewn that gouty deposits are essentially composed of crystals of sodium biurate. It will also be shewn to be extremely probable, if not certain, that the precipitation of these crystals in the joints is the actual excitant of the arthritic attacks in gout. A knowledge of the chemical relations of sodium biurate is therefore indispensable to a clear understanding of the gouty process. It is of interest to know how this compound arises in the bodily fluids, what are its relations of solubility in diverse media—especially in the blood, lymph, and synovia—and what are the factors which tend to determine or to prevent its precipitation in the tissues.

It is to be remembered that uric acid does not exist in the blood and lymph in the free state, but in a state of combination—as a urate When, therefore, uric acid is spoken of as circulating in the bodily fluids the expression is not quite exact; what really circulates is not uric acid, but a urate. There are three classes of urates which differ radically from each other both in their chemical properties and in their conditions

of origin, namely, *neutral urates*, *biurates*, and *quadriurates*. The neutral urates ($M_2\bar{u}$) are only formed when uric acid is brought into contact with alkalis in the caustic state; and, as caustic alkalis never exist in the animal body, the neutral urates can never intervene in the physiological or pathological history of uric acid. The biurates (MHU), in the form of the sodium biurate, exist, as has been seen, in gouty concretions. The quadriurates (H_2UMHU) constitute the form, and the sole form, in which uric acid subsists in normal urine. The amorphous uratic sediment of human urine consists of a mixture of the quadriurates of potassium, sodium, and ammonium in varying proportions.[1]

It may be further said that the normal or physiological status of uric acid in the body is that of a quadriurate, and that any departure from this status leads to pathological consequences. In urolithiasis the departure is towards a breaking-up of the unstable quadriurate, and a setting free of its uric acid, thereby leading to the formation of urinary gravel and calculus. In gout the departure is in the converse direction —towards the transformation of the quadriurate into the more stable biurate, and the precipitation of the latter in the tissues.

Seeing that sodium biurate is the essential component of gouty concretions, the solvent relations of this substance are of great interest in the study of the pathology of gout. The best solvent for sodium biurate is pure water, which, at blood-heat, dissolves it in the proportion of about 1 part in 1000; but if any salt of sodium be added to the water its solvent power on the biurate is greatly reduced. Water containing 0·2 per cent of the bicarbonate or chloride of sodium only dissolves about 1 part in 3000; water containing 0·5 per cent only dissolves about 1 part in 10,000; and in water containing 0·7 per cent of sodium salts the biurate is almost insoluble. All the sodium salts act alike; the carbonate and phosphate, which have an alkaline reaction, exercise just as much deterrent effect as the chloride and sulphate, which have a neutral reaction. Now the serum of the blood, and its derivatives, lymph and synovia, are very rich in sodium salts,—they contain about 0·5 per cent of sodium chloride, and about 0·2 per cent of sodium bicarbonate, making with the small amount of sodium phosphate a total of about 0·73 per cent of sodium salts; whereas the other salts of the serum—potassium, calcium, and magnesium salts—only amount to 0·11 per cent all put together. It was ascertained by direct experiment that the behaviour of uric acid and the urates with blood-serum and its derivatives depends entirely on the saline ingredients which they contain, and is quite independent of their albuminous constituents. When serum was deprived of its salts by dialysis it reacted with uric acid and the urates like simple water. It was not, therefore, surprising to find that sodium biurate is almost insoluble in serum and synovia. From a number of comparative experiments it was estimated that these fluids could not

[1] For a fuller account of the urates and the chemistry of gout, the reader is referred to Sir W. Roberts's Croonian Lectures for 1892, *On the Chemistry of Uric-Acid Gravel, and Gout*, published by Smith, Elder, and Company.

hold in solution more than about 1 part in 10,000 of crystalline sodium biurate.

Results of a corresponding character were obtained with actual uratic deposits, as shewn in the following experiments:—A metatarsal bone encrusted with uratic matter was suspended in a phial containing six ounces of blood-serum of the pig, and in fifteen months the deposit was entirely dissolved out, decomposition being prevented by the addition of a few drops of chloroform. In strong contrast with this was the behaviour with water. A second metatarsal bone from the same subject, and similarly encrusted, was suspended in six ounces of distilled water, and treated in the same way. The deposit was entirely dissolved out in four days. The cause of this immense difference was due to the large quantity of sodium salts in the serum.

Behaviour of Uric Acid and the Quadriurates with Blood-Serum and Synovia.—An examination of the reaction of free uric acid with blood-serum and synovia is a necessary part of the study of the chemistry of gout. When uric acid is digested, at the temperature of the body, with serum or synovia it passes freely into solution in combination with a base. Such a solution (when fully saturated) is found to contain as much as 1 part in 500 of uric acid. The chemical and solvent power of serum and synovia on uric acid depends entirely on the sodium carbonate they contain, which imparts to them their alkaline reaction — and has no relation to the sodium chloride and other neutral salts contained in them. The combination which is formed in these circumstances is always in the first instance a quadriurate, and not a biurate. The quadriurate is, however, an unstable salt; and, in solutions of the alkaline carbonates such as the serum of the blood, lymph, and synovia, it slowly takes up an additional atom of a base, and is thereby gradually converted into a biurate. The biurate of sodium thus formed finds itself in a medium rich in sodium salts, and one wherein, as we have seen, it is almost insoluble; it is, consequently, after a time thrown out of solution and precipitated in the crystalline form. In order to illustrate this remarkable succession of events more clearly the particulars of two experiments may be related.

Experiment 1.—Fresh serum of pig's blood was treated with uric acid in excess in a 4-oz. phial, tightly corked, and chloroformed to prevent decomposition. The phial was gently turned upside down a few times at first, but was not subsequently disturbed; it was then placed in the warm chamber. The serum soon cleared, the undissolved uric acid fell to the bottom, and the supernatant serum became transparent. For about twenty-four hours no change occurred, but in the course of the second day stars of sodium biurate were detected amid the deposit, and during the third day an abundant precipitation took place of stars and needles of biurate, exactly resembling those found in gouty concretions. On the fourth day the process of precipitation was nearly complete, and the supernatant serum was found comparatively free from uric acid.

Experiment 2.—Synovia obtained from the hip-joints of an ox was

shaken up with excess of uric acid for about half an hour, and then left at rest. In six hours the undissolved uric acid had subsided. The supernatant fluid was then carefully decanted into a 2-oz phial and placed in the warm chamber. For two days it remained quite unaltered; but on the third day precipitation of biurate began, and continued copiously during the fourth day; on the fifth day precipitation was apparently completed, and the supernatant liquor was found almost free from uric acid.

It was impossible not to be struck with a certain rough resemblance between the results observed in these experiments and the phenomena of the gouty paroxysm. In the gouty subject it is assumed that the blood becomes more and more impregnated with urates until, after a certain period of incubation has been accomplished, sudden precipitation of sodium biurate takes place in and about the joints, and the "fit of the gout" is declared. Then follows a process of recovery, with restoration of the blood to a purer state—that is, with a lessened impregnation with urates. In the artificial counterfeit we observe a similar succession of events: first, impregnation of the medium with sodium quadriurate; secondly, a period of incubation or maturation, during which the quadriurate passes gradually into biurate; thirdly, somewhat sudden precipitation of sodium biurate in the crystalline form; and, lastly, restoration of the medium to comparative purity.

There is a point of some importance in connexion with the chemistry of the biurates which may be here mentioned. The biurates are capable of existing under two distinct forms or modifications, namely, the ordinary anhydrous crystalline form seen in gouty concretions, and a hydrated gelatinous modification. The hydrated modification is much more soluble in serum and other media than the crystalline form. The hydrated condition is essentially unstable, and it tends to lapse, by a spontaneous change, into the crystalline form.

The facts above elicited respecting the chemical relations of uric acid and the urates enable us to obtain a coherent view of the history of uric acid in the living body. In normal urine uric acid always exists as a quadriurate; in animals, such as birds and serpents, which eliminate their nitrogen as uric acid, the urinary excretion is composed entirely of the same combination. It is, moreover, demonstrable that in media containing alkaline carbonates—such as the serum of the blood and its derivatives, lymph and synovia—uric acid passes into solution in the first instance as a quadriurate. From these considerations it may be inferred that in the normal state uric acid is primarily taken up in the system as a quadriurate; that it circulates in the blood as a quadriurate; and that it is finally voided with the urine as a quadriurate. In perfect health the elimination of the quadriurate proceeds with sufficient speed and completeness to prevent any undue detention or any accumulation of it in the blood. But in the gouty state this tranquil process is interrupted, either from defective action of the kidneys or from excessive introduction of urates into the circulation, and the quadriurate lingers unduly in the

blood and accumulates therein. The detained quadriurate, circulating in a medium which is rich in sodium carbonate, gradually takes up an additional atom of base, and is thereby transformed into biurate. The biurate thus produced exists at first in the hydrated or gelatinous modification; but with the lapse of time and increasing accumulation it passes on into the almost insoluble anhydrous or crystalline modification; and then precipitation of it becomes imminent, or actually takes places.

The Conditions which accelerate or retard the Processes which culminate in the Precipitation of Sodium Biurate.—Assuming a real analogy to exist between the processes which go on in serum artificially impregnated with uric acid, and the processes which go on in the blood of a gouty patient, and culminate in the deposition of uratic concretions, it is a matter of interest, as bearing on the pathology and treatment of gout, to investigate the conditions which, in the artificial parallel, accelerate or retard these processes.

As already explained, these processes consist of three distinct chemical changes. First, the quadriurate originally formed is converted into hydrated biurate; next, the hydrated biurate is changed into anhydrous biurate; and, finally, this anhydrous biurate is precipitated in the crystalline form. For the present purpose it will be more convenient to consider these changes as one continuous process, and for the sake of brevity and ease of expression this process may be designated as *maturation*.

The investigation embraced a study of the effects of temperature, percentage of uric acid in solution, and the addition of various saline and other substances to the maturating medium.

(*a*) Temperature.—It was found invariably that maturation was more quickly accomplished in the warm chamber at 100° F. than at the temperature of the room, but the ultimate result was exactly the same in both cases. For example, serum charged with 1 part of uric acid in 600 began to precipitate in the warm chamber in four hours, and precipitated copiously in six hours. A duplicate specimen kept at the temperature of the room (65° F.) began to precipitate in eight hours, and did not precipitate copiously for sixteen hours.

(*b*) Quantity of Uric Acid in Solution.—It was found that no factor exercised so great and decisive an influence on the speed of maturation and the advent of precipitation as the proportion of uric acid in solution. The copiousness of the precipitation was likewise, of course, affected by the same factor. The following experiment with blood-serum, the results of which are arranged in a tabular form, illustrates these points in a striking manner:—

[TABLE

TABLE shewing the Influence of Percentage of Uric Acid in the Medium on the Speed of Maturation and the time of Advent of Precipitation.

Quantity of Uric Acid contained in the Serum.	Time of Precipitation of Sodium Biurate.
1 in 1000 . . .	Precipitation began in six hours, copious precipitation in fourteen hours.
1 in 2000 . . .	Precipitation began in thirty-three hours, copious precipitation in three days.
1 in 3000 . . .	Slight precipitation began in three days, which became a little more copious in twelve days.
1 in 4000 . . .	A few needles of biurate were detected on the sixth day; more needles and a few tufts in twelve days.
1 in 5000 . . .	A few short needles were detected on the thirteenth day. In thirty days the needles were somewhat more numerous.
1 in 6000 . . .	No needles were discoverable in fourteen days; a few were detected in forty days.
1 in 8000 . . .	No needles could be detected after a lapse of forty days.

Assuming that the inflammatory arthritic attacks in gout are directly due to copious and sudden precipitation of crystalline stars and needles of sodic biurate in the cartilages and fibrous structures of the joints, the evidence before us indicates that such copious and sudden precipitation can only take place when the fluids bathing these structures are impregnated with uric acid in at least the proportion of 1 part in 2500. Below this point the precipitation occurs slowly and scantily, and only in the form of short scattered needles. When the proportion of uric acid dissolved in the serum was only 1 part in 5000 the deposited needles of biurate were mostly about as long as the diameter of a red blood-disc, some were twice this length, and a few three times this length; all were of extreme tenuity. It is quite conceivable that this slighter precipitation in the tissues of short scattered needles might account for certain irritations in the various organs, such as characterise irregular or larval gout, but it could scarcely engender frank inflammatory attacks. It is further conceivable that the presence in the blood of such scattered needles might constitute foci around which clotting might take place, and that the thrombosis not infrequently observed in gouty cases might be thus accounted for.

The impregnation of the blood in gouty persons with uric acid to the extent of these lesser degrees is within the range of observed actualities. Sir Alfred Garrod, by quantitative analysis, obtained from the blood-serum of one of his patients uric acid to the amount of 1 part in 5714; and he remarks that the quantity thus recoverable from the blood is probably much under the actual amount, as considerable loss may occur from unavoidable causes.

These considerations lead to the suggestion that a microscopical examination of the blood in gouty persons might sometimes reveal the existence of needles of biurate in that fluid. I tested this point in ten cases of chronic gout by examining a drop of blood drawn from the finger, but I failed to obtain positive results.

(*c*) Influence of Saline Substances.—The effect of saline substances on the maturating process was tested by adding small quantities of various salts to serum impregnated with uric acid, and observing whether these additions accelerated or retarded precipitation. The following summary indicates the conclusions deduced from the experiments:—

The addition of sodium salts to the maturating medium invariably hastened the precipitation. An idea of the degree of acceleration may be gathered from the following examples:—Serum impregnated with uric acid to the extent of 1 part per 1000 began to precipitate in seven hours, and precipitated copiously in sixteen hours. A parallel experiment, in which 0·2 per cent of sodium chloride had been added to the serum, began to precipitate in five hours, and precipitated copiously in twelve hours. Another sample of serum was impregnated with uric acid to the extent of 1 part in 2000. This began to deposit crystals in thirty hours, and deposited freely in ninety-six hours. In a parallel experiment, in which 0·2 per cent of sodium bicarbonate had been added to the medium, precipitation began in twenty hours, and free precipitation took place in forty hours. The alkaline reacting salts—the carbonate and phosphate—had exactly the same effect as the chloride and sulphate, which are neutral in reaction.

The addition of potassium salts sensibly retarded precipitation, but did not appreciably diminish the eventual amount of it. Here, again, the carbonate and phosphate, which are alkaline, produced just the same effects as the chloride, iodide, and bromide, which are neutral. Both with potassium and sodium salts the results were entirely dominated by the nature and quantity of the bases added, and had no reference to the acidulous radicle with which the bases were combined.

The addition of calcium and magnesium salts appeared to delay precipitation, but their action in this respect was quite insignificant or even doubtful. The salts of lithium had not the slightest influence either way. Piperazine, whether in the free state or as chloride, was not found to exercise any influence on the advent of precipitation.

Topography of Uratic Deposits.—The topographical distribution of uratic deposits through the various organs and tissues of the body exhibits certain well-marked characteristics. These deposits are found almost exclusively in structures belonging to the connective-tissue class—in cartilages, ligaments, tendons, and other fibrous structures, and in the cutaneous and subcutaneous connective tissues. On the other hand, uratic deposits are conspicuously absent from the muscular tissue, and from the substance of the brain, liver, spleen, and lungs. The tissues which are liable to uratic precipitations are, however, not equally so in the different parts of the body. The cartilages, ligaments, and tendons

in and about the joints, which are bathed with synovia, are much more prone to these deposits than are cartilages and fibrous structures situated at a distance from joints, and which are not bathed with synovial fluid. It is further to be noted that uratic deposits favour the more superficial and cooler parts of the body, especially the upper and lower extremities, and are more rare in the deeper and warmer internal parts of the trunk. It may be inferred from these particulars that the influences which co-operate to determine the site of uratic precipitations are of several and quite different kinds. I do not propose to discuss this subject comprehensively, but to refer to two points only which seem capable of chemical or physical elucidation. These are (*a*) the influence of the proportion of sodium salts in the several organs and tissues, and (*b*) the influence of synovia.

(*a*) Influence of the Proportion of Sodium Salts.—We have seen that the dominant factors in uratic precipitation, as studied in the laboratory, are the proportion of urates and the proportion of sodium salts contained in the medium.[1] The highest tendency to precipitation is reached when there is a concurrence of these two factors in maximum intensity. A medium may be rich in urates, but if it be at the same time poor in sodium salts, its tendency to precipitation is feeble, and vice versa. This fact has a direct bearing on the topography of uratic deposits. For if we suppose the system of a gouty man, on the eve of an outbreak, to be throughout equally impregnated with urates, it is obvious, from the experimental evidence before adduced, that uratic precipitation would take place earliest and most copiously in those parts which were richest in sodium salts, and take place latest, or not at all, in those parts which were poorest in sodium salts. Let us now examine the distribution of sodium salts in the body, and seek to ascertain if there be any correspondence between the liability to uratic deposits in the several tissues and organs and the proportion of sodium salts normally contained in them. The subjoined table shews the results of analyses on this point. The materials available for comparison are not so full and precise as could be desired, but their general significance is quite unmistakable. In order to render the comparison more complete and instructive the table includes not only the results with respect to the solid organs and tissues, but also those with respect to blood-serum and its derivatives—lymph and synovia.

TABLE shewing the Percentage of Sodium Salts in the several Fluids, Tissues, and Organs of the Body.

	Sodium Salts per cent.		Sodium Salts per cent.
Blood-serum	0·70	Blood-corpuscles	0·20
Lymph	0·70	Brain	0·20
Synovia	0·80	Muscle	0·08
Cartilage	0·90	Spleen	0·04
Fibrous tissue	0·70	Liver	0·02

[1] The proportion of calcium and magnesium salts is always too small to have any appreciable influence on the occurrence of uratic precipitation.

An inspection of the table shews that the tissues which are liable to uratic deposits are very much richer in sodium salts than the tissues and organs which are not thus liable. The remarkable immunity from uratic deposits enjoyed by the muscular tissue and by the brain, liver, and spleen may be inferred to be due, for the greater part at least, to their poverty in sodium salts. It has been shewn above that the solvent power of a medium for sodium biurate increases in correspondence with its lessening proportion of sodium salts. Brain has only about one-fourth the percentage of these salts as compared with cartilage and fibrous tissue, and muscle only one-tenth. This difference signifies (roughly) that brain has four times more power, and muscle ten times more power, of dissolving sodium biurate than cartilage and fibrous tissue, and therefore, respectively, four and ten times more power of resisting its precipitation in their substance. On this ground it might probably be truthfully said that brain, muscle, liver, and spleen could not become the sites of uratic deposits until the fibrous and cartilaginous tissues had been exhausted in this respect. It is true that the immune tissues and organs have a quicker circulation than cartilages and tendons, and this doubtless contributes importantly to the difference; but it does not fully account for it, otherwise we should expect to find that the skin, which is abundantly supplied with blood-vessels, would share this immunity. The prepotency of the cartilages and fibrous tissues to induce uratic precipitation in their substance must obviously operate in a conservative direction, and serve to protect the more vital organs of the gouty from similar precipitations, where they would produce more deadly effects.

(*b*) Influence of Synovia.—The connexion between synovia and gouty deposits is evidently very close and special. Synovial fluid itself has been repeatedly found heavily laden with crystals of sodium biurate. In the great majority of the less severe cases of gout uratic deposits are exclusively confined to those cartilages, ligaments, and tendons which are in actual contact with synovial sacs or synovial sheaths. With regard to the articular cartilages it may be demonstrated, I think, that the uratic precipitation actually takes place from the synovial fluid, and is not self-originating in the cartilaginous substance. Vertical sections of gouty cartilages examined under the microscope shew that the deposit hugs the free surface of the cartilage, and that it becomes progressively sparser towards the deeper layers—the central and deepest parts being often quite free from deposit.

This mode of distribution, moreover, implies that the process of deposition, so far as concerns the cartilage, is a purely passive and physical one, and in nowise active and vital in its initiation. We may suppose that the urate dissolved in the synovia penetrates by liquid diffusion into the superficial layers of the underlying cartilage, and that when the critical moment arrives precipitation takes place simultaneously in the synovia and in the cartilage. On this view the granular and erosive changes sometimes found in gouty cartilages are entirely secondary, and are due to the reaction of the tissue against the presence of a foreign body lodged

in its substance. It is of some interest to know that this process can be artificially imitated, and that a counterfeit gouty cartilage can be produced in the articulation of a dead animal. Samples of such counterfeits were procured in the following manner :—Tarsal bones of a pig were suspended in phials charged with a saturated solution of sodium biurate made in hot water and then cooled. The phials were chloroformed and corked, and then set aside in the warm chamber, or at a temperature of the room. Reprecipitation of the biurate took place in two or three days. If the bones were now examined, the articulating ends were found to be encrusted with a chalky matter, which could not be wiped off with a towel nor removed with a nail-brush. They presented an exquisite imitation of the plastered appearance of a gouty cartilage. If vertical sections of such cartilages (previously hardened in absolute alcohol) were made, and examined with the microscope, the deposit was seen to be situated in the substance of the tissue, close beneath the synovial surface of the cartilage, and could be identified as consisting of a dense felt of fine needles of biurate. The deeper layers of the cartilage were not affected.

It may be inferred that the uratic deposits which occur in the ligamentous and tendinous structures of gouty joints are produced in the same way as those found in the articular cartilages ; that the precipitations take place, chiefly at least, from the synovial fluid with which they are bathed, and that they are not primarily due to morbid changes in the structure of the tissues.

Two questions may now be asked :—First, why does gouty precipitation take place preferentially in synovia rather than in its cognates, the serum of the blood, and lymph ? And, second, why do the joints differ so much from each other in their liability to attack ? I will discuss the two questions together, as the arguments often dovetail into each other. It may be that there is, as Sir A. Garrod suggests, some special attraction in the joints for uric acid. But there are undoubtedly other factors which come into play. Synovia is a comparatively motionless fluid, while serum and lymph are in ceaseless motion. And as a still pool crystallises into ice sooner than a running brook, so likewise—supposing serum, lymph, and synovia to be equally impregnated with urates and sodium salts—the tranquillity prevailing within the synovial sacs would give to synovia a priority in uratic precipitation over the restless blood-serum and lymph. Then, as regards the varying liability of the different joints to gouty attacks, it may be pointed out that the synovial pouches and sheaths are shut sacs, standing apart from each other, and isolated from the general panmixia of the circulation. It might consequently be expected that the synovial fluids in different joints should present differences in the relative proportions of their constituents ; indeed, this is so. Some are certainly more concentrated than others, and we can easily believe that they are not quite identical in the degree of their impregnation with urates and with sodium salts. In this way the several joints might come to vary considerably in their liability to uratic precipitation.

Frerichs has contributed some interesting particulars on synovia in animals. His observations indicate that synovia varies both in quantity and quality under different modes of life. He found that stall-fed horses and oxen, leading an idle existence, had twice as much synovia in their joints as similar animals roaming in the meadows or doing work. Moreover, the composition of the fluid varied in the contrasted conditions. In the idle animals the synovia was more watery, and contained less albuminoid matters, but—and this is significant—a larger proportion of mineral salts, which consist almost entirely of sodium salts. We might conjecture from this observation that, if horses and oxen were liable to uratic precipitations, the idle stall-fed animals would be more subject to such deposits than the same animals leading a more active life. Perhaps we may discern herein one reason why men who lead a sedentary life are more subject to gouty deposits than men who take active exercise.

Interpretation of the Appearances found at the Necropsy of Gouty Subjects. Re-solution of Gouty Deposits.—Before leaving the subject of the topographical distribution of gouty deposits, a word may be said on the need of caution in interpreting the appearances, both positive and negative, observed at the necropsy of gouty subjects. The incidents of the gouty diathesis, especially in the earlier periods, pursue a markedly interrupted course. Long intervals of months or years often elapse between the arthritic outbreaks. During these intervals the blood of the gouty man recovers its purity more or less completely, and approximates in its content of uric acid to the blood of a healthy person. The solvent relation of the bodily fluids to the material of gouty deposits is simply a question of saturation or subsaturation. If the lymph or synovia at a certain spot become saturated, or rather supersaturated, with sodium biurate, precipitation of that substance will inevitably take place into the contiguous fibrous tissues; and if, after such an event, the lymph or synovia recover its purity and become approximately free from biurate, as in due course usually comes about, a process of slow re-solution will of necessity set in. For, as has been before demonstrated, sodium biurate, although very sparingly soluble, is not absolutely insoluble in these media. The rate and amount of re-solution of uratic deposits must necessarily vary greatly in different cases—according to the degree of subsaturation attained by the bodily fluids, the massiveness and penetrability of the deposits, and the length of time during which the favourable conditions endure.

If regard be had to the, often, long survivorship of gouty persons, and the interrupted course of the arthritic incidents, it seems highly probable, in the lapse of a long life, that deposition and re-solution of uratic matter may take place once and again in a gouty joint. There are undoubted instances, as has been already pointed out, in which no uratic deposits have been found after death in joints which, at some previous period, had undergone definite gouty attacks. The presumption in such a case is, not that deposits never existed, but that in the intervals of amendment they had been redissolved. Uratic concretions in the pinna of the

ear have sometimes been actually observed to come and go, and come again. There is no reason why the same thing should not occur within the joints; and such vanished concretions might, or might not, leave behind them permanent changes in the cartilages and bony structures as tell-tale evidence of their former presence. On the other hand, the discovery after death of uratic deposits in a joint is not always to be regarded as a certain proof that the joint had passed through an inflammatory gouty attack. The observations of Moxon and Fagge seem to warrant this conclusion; and they reasonably suggest that when the precipitation of the urates takes place slowly and by degrees, there may be no accompanying inflammatory outbreak to mark the event (4). The cases of Levison, before cited, also support this conclusion (see p. 129).

The experimental evidence before adduced indicates that when lymph or synovia is impregnated with sodium biurate to or above 1 part in 6000, the medium is supersaturated, and precipitation either actually occurs or is impending. On the other hand, when these fluids contain less biurate than 1 part in 10,000, the medium is undersaturated, and there resides in it a certain power of redissolving uratic deposits; the nearer the medium is to freedom from urates, the higher, of course, rises this solvent power. It may, further, be inferred that the solvent action will be most effective in the case of deposits situated in textures like the fibrous tissues, which have a comparatively free lymph flow; and, conversely, that it will be least effective in textures like the cartilages, which have a sluggish lymph flow. This is probably the reason, or the chief reason, why the cartilages figure more prominently than the fibrous structures in the morbid anatomy of old gouty joints. Probably both tissues were originally infiltrated with biurate crystals in equal degrees; but the fibrous structures afford greater facilities for their re-solution in the periods of amendment than do the cartilages, hence the greater persistence of the deposits in the latter tissue.

The Mode in which Uric Acid produces its Injurious Effects.—A problem of great interest in the elucidation of gouty manifestations is the mode or modes in which uric acid produces its injurious effects. The main question is, whether these effects are exclusively due to the *mechanical damage* consequent on its precipitation as sodium biurate in the tissues; or whether, in addition, uric acid circulating in the blood in a state of solution is capable of acting as a *chemical poison*.

With regard to the incidents of regular gout, the mechanical hypothesis seems to offer a natural and complete explanation. The crystals of sodium biurate precipitated in the cartilaginous and fibrous structures of the joints necessarily act as foreign bodies; they excite irritation, clog the lymph-channels, exercise pressure on the tissue-elements, and impede their nutritive operations. These effects sufficiently account for the inflammation, pain, and swelling which ensue, and explain the remoter degenerative changes which follow after. Nor need we look beyond physical conditions to account for the diversity of the local manifestations. It is easy to understand that depositions occurring within the tense, unyielding

structures of the joints would produce results widely different from similar depositions in the loose subcutaneous tissue, or in the rim of the ear. It is equally easy to understand that the suddenness or slowness of the precipitation, its copiousness or scantiness, would necessarily cause great variation in the intensity and character of the local disturbances. So easy and natural is this explanation, that from our general knowledge of pathological cause and effect we might even predict that if similar sudden and copious depositions of crystals of carbonate of lime, or of any other inert substance, were to take place in the same localities, there would follow very much the same train of morbid sequences as are witnessed in connexion with uratic precipitations.

It is to the explanation of the phenomena of irregular gout that the mechanical conception of the action of uric acid seems inadequate; and it is to meet this lack that the hypothesis of a poisonous action has been set up and is invoked. The visceral disturbances and manifold neuroses which trouble the gouty have not yet been anatomically traced to uratic precipitation, and there seemed no other way of explaining their occurrence—if they were to be linked with uric acid at all—except by assuming uric acid—or rather the urates—to be possessed of toxic properties. The acceptance of this view appears on several grounds to be extremely difficult.

There is, first, complete absence of direct experimental proof that uric acid is poisonous. Animals have been made to ingest large quantities of uric acid with their food, and urates in solution have been freely injected into their veins, without eliciting any signs of poisoning.

In the next place, the notion that uric acid is poisonous seems opposed to broad biological analogies. Uric acid is the physiological homologue of urea; each of these bodies constitutes, in its separate domain, the final term of nitrogenous metabolism. It cannot be said, without an abuse of terms, that urea is a poisonous substance; and it would be strange if its homologue, uric acid, differed from it in so important a particular as the possession of toxic properties.

The hypothesis appears not less improbable when examined from a nearer point of view. The system of the gouty man is at times surcharged with urates. On the eve of an outbreak the fluids of his body, in parts at least, must be impregnated with biurates to saturation; for, of course, no precipitation can occur until this point is reached. Yet, with fluids thus saturated with urates, such persons often betray not the slightest sign of poisoning, and enjoy complete immunity from symptoms of every kind until overtaken, unwarned, by the sudden precipitation which provokes the arthritic attack.

Again, the manifestations of irregular gout are so extremely diverse in seat and character that it is hard to believe that they can be produced by one and the same toxic agent. Sometimes they implicate the stomach, sometimes the liver, heart, or lungs, and, oftenest of all, the nervous system. This diversity is, however, easily explicable on the supposition that the disturbances are caused, not by uric acid in a state of solution

acting as a poison, but, like the arthritic manifestations, by uratic deposition; that is to say, by actual precipitation of crystals of biurate into the connective and fibrous structures of the implicated organs, or into the fibrous sheaths of the nerves which control their functions. Observations at the bedside and in the dead-house lead to the inference that uratic precipitation is very variable in its mode and incidence. In certain conditions the crystals appear to descend in sudden and copious showers, which, as in the regular arthritic seizures, provoke a sharp inflammatory reaction. Under other conditions the crystals seem to fall in gentle sprinklings, sufficient perhaps to cause irritation if the implicated tissue be a sensitive one, but not enough to cause downright inflammation. The peculiar pricking pains in the joints which some gouty persons invariably experience after partaking of certain wines are highly suggestive of the occurrence of these slighter precipitations. Now if these slighter precipitations, instead of falling on the joints, were to fall upon the membranes of the brain or upon the fibrous sheaths of the nerve-roots, they would, I submit, afford an adequate explanation of the phenomena of irregular gout. Of course it may be objected that no such slight precipitations have actually been found. But have they been looked for? Has the microscope been used in the investigation? In prosecuting such a search it would have to be borne in mind that precipitations of the kind supposed would be apt to be fugitive, and that negative results would have to be interpreted with caution. For it is obvious, in the case supposed, that when the stress of saturation of the fluids with urates was relaxed, and the blood again recovered its power of dissolving these compounds, these slight deposits would be speedily removed by re-solution, and not a trace of them might remain at the autopsy.

We scarcely realise how imminent a slight but widespread precipitation of the crystalline biurate must not infrequently be in the gouty system—implicating the blood and lymph, as well as the fibrous tissues throughout the body. It has been already shewn that when the serum of the blood is impregnated with sodium biurate to the extent of 1 part in 6000, supersaturation is attained; and precipitation is then, of course, imminent: now Sir A. Garrod has proved by quantitative analysis that the blood-serum of the gouty man is sometimes actually impregnated with uric acid to this extent. These two facts taken together indicate that the explanation of the phenomena of irregular gout here suggested stands on a strong basis of a priori probability—and thus dispenses with the necessity of assuming that uric acid and its compounds are endowed with poisonous qualities.

This mode of viewing the subject enables us to bring the diverse morbid effects of uric acid into uniform line. Uric acid and its compounds are deleterious simply because of their sparing solubility in the bodily media. It may be said that the final cause of uric-acid gravel is the sparing solubility of free uric acid in urine; in like manner it may be said that the final cause of gouty precipitations is the sparing solubility of sodium biurate in blood-serum, lymph, and synovia.

The facts and arguments adduced by the late Sir William Roberts with regard to the relation of urataemia and uratosis to gout are of much weight, but like other hypotheses of gout present considerable difficulties, and have met with opposition and criticism. In the first place, some doubt has been thrown on the existence of quadriurates as definite compounds, and also on the question whether uric acid exists in the blood as a quadriurate. Analyses have apparently shewn that the so-called quadriurates of the urine have not always the composition required by the formula $MHUH_2U$, an excess of bases is frequently present, and thus these observers have regarded the deposit as a mixture of uric acid and biurates in varying proportions (Tunnicliffe and Rosenheim). To meet this objection it has been suggested that the so-called quadriurates may have a greater number of molecules of uric acid loosely combined with biurates and with the formula $MHU.nH_2U$ instead of that assigned by Roberts. The experiments carried out by Roberts would seem to shew that uric acid is dissolved in the blood in some other form than that of a biurate, since the solubility of this salt is insufficient to account for the quantities of uric acid that can be dissolved in serum. Difficulties such as these have led to the suggestion that uric acid is perhaps normally dissolved in the blood in the form of an organic compound from which the kidney is able to split off uric acid. This at present is only a speculation, but if correct might have an important bearing on the nature of urataemia and of gout, since it is possible that in gout uric acid might be present in the blood in an abnormal form as well as in excess, or even that in some instances there might be no excess but only the abnormal form. Such a view might explain the absence of excess of uric acid in the blood of the gouty described by some observers.

The condition of urataemia is one of much importance in the pathology of gout, and is necessarily closely associated with the physiology of uric acid. Uric acid is a diureid, that is, its molecule contains two urea radicles joined together by a tri-carbon group, and although uric acid can be broken up into urea its more important relationship is with the purin bases. Purin has the empirical formula $C_5H_4N_4$, and uric acid is an oxypurin. The other oxypurins met with in the body and in the food are xanthine and hypoxanthine. Adenine and guanine are purins in which the oxygen atom is replaced by an amido group; and these enter into the composition of nuclein. The decomposition of nuclein in the body leads to the formation of xanthine and hypoxanthine, and these are in part oxidised to uric acid and excreted as such.

The uric acid excreted in the urine has a twofold origin, part being derived from the food and part directly from the metabolism of the tissues. The former is often spoken of as exogenous and the latter as endogenous in origin. At one time it was thought that the quantity of uric acid in the urine was mainly dependent on the quantity of protein in the food as is the case with urea, and that a certain definite relationship existed between the quantities of these two substances excreted, and further, that the formation of uric acid might be looked

upon as the result of the deficient oxidation of proteins. The exogenous uric acid, however, is not related to the *quantity* of protein ingested, but to the *nature* of the protein, in other words, it depends on the ingestion of purin bases. By the administration of a diet free from purin bases the uric acid in the urine falls to a certain constant quantity for each individual, and all is then derived from the metabolism of the tissues. Roughly, about one-half of the uric acid normally present in the urine arises from the purins in the food, and the other half from the purins metabolised from the tissues. Although the uric acid in the urine is derived from the purins of the food and tissues, yet the whole of the purins of the food and probably also those of the tissues are not converted into uric acid. In the case of such substances as hypoxanthine present in the food about one-half appears in the urine as uric acid, the other half being broken up in the course of metabolism into urea, carbon dioxide, and water.

These conclusions have largely been deduced from the experiments of Burian and Schur, who studied the excretion of purins under different diets. In their experiments four different diets were investigated. First, a meat diet containing a large quantity of nitrogen. Secondly, a milk, cheese, and egg diet containing the same quantity of nitrogen. Thirdly, a milk, cheese, and egg diet containing much less nitrogen than diet No. 2, owing to rice being substituted for a portion of the milk, cheese, and eggs. Fourthly, a vegetable diet containing the same amount of nitrogen as diet No. 3. The examination of the urine shewed that the quantity of uric acid and other purins was greatest in the flesh diet; but that the quantities excreted with the other three diets were approximately equal notwithstanding the very great differences in the amount of protein contained in these several diets. The flesh diet contained, of course, considerable quantities of nuclein and other purin bases, whereas the other three diets, although varying in their nitrogen content, all resembled one another in being practically free from purin bases.

These experiments shewed that the uric acid excretion of endogenous origin was uninfluenced by the diet so long as this was free from purins. The amount of nitrogen excreted in the form of purin bodies on a purin-free diet, *i.e.* the endogenous purin-nitrogen, is remarkably constant for the same individual, although it may vary within wide limits in different individuals. The variation in different individuals is of obscure origin, and does not seem to be related to the body-weight nor even to the amount of work that is performed. The exogenous purin-nitrogen derived from the metabolism of the food owes its origin to the purin bodies present in glands and muscles. Xanthine and hypoxanthine and nuclein are the most important of these, and speaking roughly about one-half of the nitrogen of hypoxanthine reappears in the urine as uric acid, and this fraction is constant in all the individuals of the same species.

Some controversy has taken place with reference to the destination of the other half of the nitrogen of the purin bases of the food. Some writers have thought that this portion becomes stored up somewhere in

the organism; others have thought that it is destroyed. Numerous experiments have shewn that the extract of the liver in certain animals, especially the dog, is capable of destroying uric acid by oxidation. In other animals, especially the herbivora, extract of kidney is capable of decomposing uric acid. The oxidation of uric acid by the liver probably explains why uric acid does not accumulate in the blood after double nephrectomy. That some of the purin bodies may be oxidised during the process of metabolism by such organs as the liver may be of considerable importance in the pathology of uric acid.

The main facts derived from experimental investigation may shortly be summarised by stating that the uric acid normally excreted is of double origin, part being derived from the purin bases in the food and part from the metabolism of the tissues, especially probably the muscles; that approximately half the purin-nitrogen of the purin bases of the food is excreted in the form of uric acid, etc., in the urine, and that the remaining portion undergoes oxidation in the body, especially in the liver, and that normally the blood is kept free from appreciable quantities of uric acid partly owing to the excretion by the kidney and partly owing to this oxidation. Lastly, that the quantity of nitrogen in the urine of endogenous origin, and therefore derived from the metabolism of the tissues, is constant for one and the same individual, but varies within wide limits in different individuals.

Granting that in some cases of gout, if not in all, an excess of uric acid may be present in the blood, this excess may arise from one of three causes: (1) an excessive ingestion of purin bases in the food; (2) a diminished destruction of purin bases in the tissues, especially the liver; and (3) a diminished excretion of such bases by the kidneys. There can be no doubt that a diminished renal excretion of uric acid is very often present in gout, and this theoretically may arise either from inability on the part of the kidney to excrete uric acid, or else, as suggested by von Noorden and others, that the compound of uric acid with some organic substance that is normally present in the blood is not formed, and thus the uric acid reaches the kidney in an abnormal form that is excreted with difficulty.

It is probable that the mere excessive ingestion of purin bases and the diminution in the oxidation of purin bases in the tissues would scarcely lead to a condition of urataemia if the excretory activity of the kidney were unimpaired. The normal kidney can excrete very large quantities of uric acid, for example in leukaemia.

The topographical distribution of uratic deposits in gout may be very well explained by Roberts's observations on the influence of sodium salts in determining the precipitation of biurates, but this hypothesis scarcely explains the whole problem of uratosis since it fails to account for the absence of uratic deposits in the joints and fibrous tissue in other non-gouty conditions in which an excess of uric acid is present in the blood. It may be that in the non-gouty states, where an excess of uric acid is present in the blood, the renal activity is able to remove the excess with sufficient rapidity to prevent the deposition of the biurates, but this

absence of deposits in non-gouty states has also been used in support of the argument that the essential point in gout is not so much an excess of uric acid in the blood as its presence in an abnormal form.

Symptoms and Course of Regular Gout.—The first invasion of gout is usually unexpected. A middle-aged man, in the midst of apparent health, is suddenly seized in the night or early morning with pain in the ball of one of the great toes implicating the metatarso-phalangeal joint. By morning the joint is red, tense, swollen, immovable, and exquisitely tender. These symptoms are accompanied by fever, scanty, high-coloured sedimentary urine, parched tongue, thirst, and anorexia. The patient is unable to put any weight on the ailing foot, and he is forced to remain in bed, or at least to keep his room. In the course of the day succeeding the onset there is usually considerable remission of the symptoms; but with the advent of evening these return in aggravated intensity, and the second night of the attack is restless, sleepless, and marked by severe pain and rising fever. In the course of the second and third days the swelling about the joint increases, and as this goes on the tension and pain diminish, and the fever tends to subside. In a week or ten days the paroxysm exhausts itself; the tumefaction, stiffness, and tenderness pass away, and health is restored. Such is often the history of a first "fit of the gout." Not infrequently, however, the mischief is not confined to the ball of the great toe—other joints are implicated: the tarsal and metatarsal joints partake in the disturbance, and the joints of the opposite foot are affected, either at the same time or in quick succession. The paroxysm is then of longer duration, and extends to two or three weeks before final subsidence. The recovery from the first attack of gout is usually speedy and complete—the joints regain their normal size and suppleness, and the general health is perfectly re-established.

After the initial attack of acute sthenic gout, such as that above described, there is often an interval of one, two, or three years before another visitation. But the tendency to recurrence usually becomes more and more pronounced as years go by; and the gouty man at length finds himself liable to an attack once a year, or twice a year, in the spring and autumn seasons, with some approach to periodic regularity. As the disease fastens on the constitution a larger number of joints are attacked; the knees become affected, the joints of the hands, and the wrists and elbows; the attacks assume a subacute character, and the condition called chronic gout is entered upon. Recovery from the paroxysms then becomes less complete, the recurrences more frequent, and the disturbance more lingering and persistent; some of the joints become permanently enlarged, stiffened, and deformed, and chalk-stones make their appearance on the knuckles and toes, and about the knees and elbows. If the disease proceed unchecked life becomes an almost continuous martyrdom, and the constitution is seriously impaired. Gout, however, is not often the direct and immediate cause of death. More frequently it kills by one of its complications—secondary inflammation, chronic Bright's disease, embolism,

thrombosis, or arterial degeneration. Fortunately it is only exceptionally that gout proceeds in this inveterate fashion: more commonly, under the influence of change of habits aided by medical treatment, the downward course is arrested or mitigated; the recurrence of the attacks is diminished, and their severity and duration lessened. It is often the case that as years advance the gouty manifestations tend to decrease; and not infrequently persons, who during the middle periods of life have suffered severely from gout, attain in old age to a state of good, or at least fair health; their gout is either greatly subjugated or has entirely ceased to trouble them.

Some of the features of regular gout above sketched require more detailed consideration. The *recurrence of gouty paroxysms* exhibits great uncertainty. The disease sometimes exhausts itself in a single paroxysm; or the second attack may only occur after the lapse of decades. Thus, a Yorkshire squire who had a characteristic seizure of gout at the age of twenty-seven, had his next attack in his eighty-ninth year. In the milder forms of gout it is by no means uncommon to find that the history of the disease is comprised in the incidents of two or three or half a dozen attacks, scattered irregularly over a long life; or perhaps compressed into the ten years of middle age. The more common rule, however, is for attacks to occur once a year or twice a year. Even in these ordinary examples breaks in the regularity of the periodicity constantly occur: either an attack comes before its time, or is postponed, or altogether omitted; or an intermediate visitation happens. In very chronic intractable cases all regularity is lost, and one attack almost runs into another; or the attacks are suspended for two or three years and then recur again as beforetime. These irregularities and vicissitudes are often quite inexplicable; in many instances they are dependent neither upon medical treatment nor upon altered dietetic habits, but are due to spontaneous changes in the constitution. They form part of the natural history of gout; and it is important to bear their existence in mind when we seek to estimate the value of therapeutic means, in order to prevent ourselves from becoming the dupes of misinterpreted sequences.

Premonitory Signs.—A gouty man often has premonitions which warn him that a seizure is impending. These warning signs may prevail for many days, or even weeks before the actual breaking out of the attack. They are exceedingly diverse. The most common are digestive troubles —loss of appetite, flatulence, and acidity—with irregular action of the bowels, and the passage of remarkably offensive motions. Very often the most prominent signs are various nervous disorders—cramps in the legs, restlessness and irritability of temper, neuralgia, pricking pain in the joints, irritating cough, unaccountable depression of spirits, persistent weariness, headache, and palpitation of the heart. All these symptoms disappear at once with the onset of the arthritic attack. But it frequently happens that no warning is sounded: the seizure comes on unexpectedly, and is a disappointing surprise to the patient, who perhaps

at the time was feeling unusually well, and had no presentiment whatever of the coming storm.

The *fever* which accompanies a gouty paroxysm is not usually high; it varies in degree with the severity of the local inflammation and the number of joints affected. The appearance of an inflamed gouty joint is peculiar, and suggestive of the reigning diathesis. The joint is at first tumid, tense, shining, red, and very tender, and the cutaneous veins near it are enlarged. As the swelling increases the pain lessens; later the swollen parts pit on pressure, and, finally, desquamate as resolution is achieved. Suppuration does not attend on ordinary gouty inflammation; but when the larger joints and bursae are implicated there is often copious effusion of serous fluid into their cavities.

Joints Affected.—Gouty inflammation has a characteristic tendency to attack the metatarso-phalangeal articulations of the great toes. These joints rarely escape, and often they are the only joints implicated. Of 516 cases of gout collated by Scudamore the local manifestations were confined in no less than 341 instances to one or both great toes. The next most frequently attacked joints are those of the ankle, instep, fingers, wrists, and knees. The elbow is much less frequently affected; the hips, shoulders, the articulation of the jaw, and the vertebral joints are but rarely involved. Joints which have been damaged by some previous accident or disease are more prone to take on gouty inflammation than sound joints.

Uratic Deposits (*Chalk-Stones, Tophi, Gouty Concretions*).—The occurrence of deposits of urates in the tissues of the body is the most characteristic feature of the gouty state. When a joint is the seat of gouty inflammation uratic deposits take place in its cartilages and fibrous structures. Such deposits, when confined to the interior of the joints, are of course not clinically recognisable. But, as has been shewn, there is reason to believe that they are an invariable incident of an arthritic attack, and are moreover the actual excitant of the inflammatory process.

In recurrent long-continued gout these deposits are not confined to the interior of the articulations. They take place, also, in the parts outside and around the joints—in the ligaments, tendons, and subcutaneous tissue; they also invade the tendinous sheaths and synovial bursae in the vicinity of joints, and thereby cause enlargements, thickenings, cripplings, and deformities of the implicated limbs. They then become a prominent feature of the clinical portraiture of gout, and constitute the source of the chief symptoms and physical signs of the complaint. Gouty concretions (or tophi) are essentially composed of sodium biurate disseminated, in felted masses of crystalline needles, through a matrix of connective tissue. In this way are produced the knobby, bulbous fingers and the distorted toes and knees of gouty persons. In old-standing cases gouty concretions may be enormous. Masses as large as walnuts, and even as large as small oranges, are now and then seen in the neighbourhood of the elbows and knees. When the deposits approach the surface they

appear as yellowish-white masses through the skin, and occasionally they push through the cutaneous covering and form indolent ulcers over the knuckles, finger-joints, or toes, and discharge a purulent ichor containing myriads of needles of sodium biurate.

Uratic concretions are not restricted to the vicinity of joints and synovial sacs. They are found in the cutaneous and subcutaneous tissues of the palms of the hands and the soles of the feet, in the penis, in the sclerotic coat of the eye, and in the superficial aponeuroses of the limbs. The most frequent seat of abarticular gouty concretions is, however, the skin of the ear. Sir A. Garrod gives the following interesting description of their formation in this situation :—"The earliest appearance presented is that of a small vesicle under the skin of the helix, as if situated between it and the fibro-cartilage. The contents of the vesicle are at first opalescent or milky, but afterwards become white and opaque, and acquire the consistence of cream. After some months the vesicle assumes the appearance of a small hard and white bead, closely resembling a pearl, and it may remain as such for years; but occasionally the thin skin is worn off, and the bead itself becomes detached from the cartilage, leaving only a slight indication of its presence. If the vesicle is punctured in the early stage a milky fluid exudes, which presents under the microscope the appearance of a transparent liquid, in which are floating a large number of very fine crystalline needles. If the contents are examined at a later stage the crystals are found aggregated into small bundles; if the bead is solid it is difficult to separate them, as they adhere strongly together, and form a closely interlaced crystalline mass." The existence of visible uratic deposits is far from being constant even in long-standing cases of recurrent gout; but when discovered they constitute an invaluable diagnostic mark. The number and extent of the visible uratic deposits in a gouty man present every degree of variation.

The severity of the associated symptoms is by no means proportional to the extent of the uratic deposits. Patients with numerous and enormous chalk-stones often suffer comparatively little. Other patients with few or no visible concretions suffer greatly. It would appear as if the irritation and inflammatory disturbance caused by uratic deposits depended more on their site than on their extent. Even slight deposits taking place in the interior of joints may cause acute suffering; but immense deposits may accumulate outside the joints, or in detached bursae, without provoking symptoms. The pearly concretions on the ears of the gouty rarely attract the attention of the patients.

Irregular Gout.—In the intervals between the arthritic paroxysms gouty persons, especially in the earlier periods of the complaint, often enjoy perfect health, and are free from symptoms of any kind; but this is not always the case. In chronic and long-continued cases, in which repeated attacks have occurred, the gouty man is well aware that his enemy is not altogether dormant even during the intervals; and he is troubled more or less with certain disturbances and symptoms which he

recognises as gouty. These disturbances go by the name of irregular or suppressed gout; they are most varied in character, and may implicate any of the functions or systems of the body. They are sometimes premonitory of an attack of regular gout, but often they come and go without reference to the occurrence of any arthritic seizure. Symptoms of a similar or kindred character may shew themselves in persons who have never had any regular articular paroxysms, and who do not present any visible uratic deposits. In cases of this class the connexion of the symptoms with the gouty diathesis is often not a matter of diagnostic certainty, but rather an inference from their otherwise inexplicable origin, or from the existence of a gouty family history.

Retrocedent Gout.—The inflammation in a gouty joint sometimes subsides suddenly, either from some unknown cause, or as the result of exposure and chill, or of the imprudent application of cold to the joints. Such sudden subsidence of the local manifestation is liable to be followed by alarming symptoms in some internal organ—the brain, stomach, or heart. This kind of metastasis is attended with grave danger to life.

Gouty Affections of the Skin.—Cutaneous affections are frequently seen in the gouty, and their direct relation to the diathesis is quite undoubted. The most distinctive is eczema. The favourite seat of gouty eczema is the external ear and the parts around, namely, the face, forehead, and the back of the neck. As a rule gouty eczema is not severe, although very persistent, and is mostly confined to the parts mentioned. Sometimes, however, it becomes a grave trouble, especially in persons of advanced years, and spreads over a large part of the body. Patches of psoriasis or dry scaly eczema, fixed, circumscribed, and often asymmetrical, sometimes appear on the legs and elsewhere. Pruritus, either local or more general, is not uncommon, and is occasionally troublesome, particularly at night. The nails of gouty people sometimes give trouble; they become ribbed and brittle, and difficult to keep in trim order.

Gouty Affections of the Eye.—Various affections of the eye have been observed in gouty persons. The most common are conjunctivitis and scleritis. In two such cases Sir A. Garrod detected uratic deposits in the sclerotic coat. Gouty iritis and glaucoma have also been described. Mr. Jonathan Hutchinson has called attention to the occurrence of haemorrhagic retinitis in connexion with gout. This condition comes on suddenly, and is always unilateral; it is probably occasioned by venous obstruction and thrombosis of the retinal vein.

Gouty Affections of the Nervous System.—The nervous disturbances observed in gout include recurrent headache, persistent depression of spirits, and various forms of neuralgia or neuritis. Among the latter sciatica is one of the commonest. Facial neuralgia is sometimes exceedingly troublesome; it interferes with the ingestion and mastication of food, and the patient may be obliged to have all his food finely cut up for a time, or he may even be reduced to spoon-meat. In other cases the neuralgic pains affect the abdominal viscera, or shift irregularly from

place to place. In rarer instances the nervous disturbances assume a more serious form and go on to delirium, insanity, or epilepsy.

The circulatory system is affected in gout in various ways. Palpitation of the heart with faltering, intermittent pulse, and syncopal threatenings are not infrequent. A very peculiar paroxysmal disturbance of the circulation is now and then witnessed, in which the cardiac pulsations become extremely rapid; and a condition is produced which may be termed a "runaway heart." Paroxysms of this kind are very alarming and compel the patient for a time to lie down. Sometimes the disturbance assumes the features of angina pectoris, and constitutes a "false angina," which may be symptomatically indistinguishable from true angina; cases of this kind, which are unassociated with organic lesions of the heart, are to be carefully distinguished from cases in which true angina occurs as a complication of the gouty state. There is no direct connexion between gout and pericarditis or endocarditis—in which respect gout differs conspicuously from rheumatic fever. When, however, the kidneys are diseased and the gouty form of Bright's disease is developed, there is, as in other forms of granular kidney, a liability to pericarditis of very fatal tendency. Gouty persons exhibit a marked proclivity to thrombosis in the extremities, most frequently in the lower, and pulmonary embolism is not an uncommon fatal complication. When this tendency exists there is liability to apoplectic seizures from cerebral thrombosis.

The respiratory organs not infrequently feel the influence of the diathesis; and gouty persons are liable to pneumonia, bronchitis, and asthmatic paroxysms.

The Urine in Gout.—The state of the urine in gout varies greatly according to the acute or chronic phase of the complaint, and according as the disease is or is not complicated with organic changes in the kidneys. During paroxysms of acute gout, occurring in otherwise healthy persons, the urine presents the usual febrile features: it is high-coloured, acid, and scanty; and on standing and cooling it deposits an abundant sediment of pink or brick-dust urates. If the fever run high the urine may contain a trace of albumin; but not more, nor oftener, than in equivalent pyrexia from other causes. The copious deposits of amorphous urates during acute attacks of gout have led to the belief that there is an excessive excretion of uric acid at these periods; but this is not really the case. The analyses of Sir A. Garrod indicate that in the early days of an attack the daily excretion of uric acid falls rather below the normal average. As the attack subsides, indeed, there is some increase, but this increase is not peculiar to gouty inflammation; it partakes rather of the nature of the critical discharges of uric acid, which are common to commencing resolution in all forms of acute inflammation.

Much more interest attaches to the state of the urine in the intervals between the gouty paroxysms than during the attack itself, when the local inflammations and the associated pyrexia mask the influence of the reigning diathesis. The most trustworthy information on this subject has been supplied by Sir. A. Garrod. He divides the cases examined by

him into two groups. The first group consisted of seventeen cases of chronic gout, in the majority of which there were no very urgent symptoms, but in which such sequels of the disease occurred as uratic concretions in different parts of the body, and stiffened and deformed joints. In these seventeen cases the urine was carefully analysed over a series of days. In all of them the daily discharge of uric acid was greatly below the normal average, which may be taken to be about 8 grains per day. The highest recorded daily discharge was only 5·78 grains, and that only on a single day. The average for the whole number stood under one grain per day. In several instances the total quantity of uric acid in the day's urine was so small that it could not be weighed; and sometimes the addition of an acid failed to shew even a trace of uric acid in the urine. As a rule the urine in this group of cases was copious, of low density, and paler than in the healthy state; and in more than half the cases a small amount of albumin was present in the urine. The second group consisted of six persons who were subject to occasional attacks of gout, but who were, at the time of the examination, completely free from symptoms. In none of this group was there any albumin in the urine. The daily excretion of uric acid in all of them was found to be below the normal mean; in some it scarcely exceeded one-third or one-sixth of the usual quantity. In both these groups of cases, as well as in cases of acute gout, the daily excretion of urea did not shew any appreciable deviation from the normal. Most recent observers have confirmed, with the aid of modern and more accurate methods, the statement that in the intervals between attacks the uric acid output may be much diminished. Further, on the second or third day after an acute attack, the daily excretion may not only reach the normal but may exceed it. Some authorities have stated that variations in the amount of phosphoric acid in the urine occur of a similar character to the variations in the uric acid. In other words, that the curves illustrating the daily output of phosphoric and uric acid are similarly affected in gout. This, however, has been denied by others and would therefore not seem to be constant.

In some cases of chronic gout there is a pronounced tendency to *uric-acid gravel*. This tendency is shewn either in the deposition of free uric acid in the freshly voided urine, or in the formation of concretions of uric acid in the precincts of the kidneys, causing attacks of renal colic followed by the passage of calculi along the ureters. These manifestations often assume a paroxysmal character, and the paroxysms of gravel appear to alternate roughly with the arthritic paroxysms. Seldom or almost never do paroxysms of gravel coincide with the arthritic paroxysms. It would at first sight appear paradoxical that uric-acid gravel should occur in chronic gout, where, as we have seen, the excretion of uric acid is diminished. The explanation of the apparent contradiction lies in the fact that in the interparoxysmal periods of chronic gout the urine is, in many cases, persistently of low density and almost devoid of pigment, a condition which, as I have elsewhere shewn, is favourable to the deposition

of free uric acid, even when the percentage of that substance in the urine is below the normal standard.

It is not to be assumed from the foregoing observations that a defective excretion of uric acid is an invariable feature of the gouty diathesis. A number of published analyses shew that gouty persons may void uric acid at the full average rate, or even above that rate; and it is within the knowledge of every experienced clinical observer that men who are undoubtedly gouty, and who have gone through typical attacks of gout, may pass a urine which contains its full complement of uric acid, and is in all respects perfectly normal. Such cases are not difficult to explain. In the first place, the excretion rate of uric acid is a variable one; it varies with what may be called the physiological idiosyncrasy of the individual; and an excretion rate which is equal to the general average may be a defective rate for a particular individual. Again, a gouty man is not at all times actively gouty. The diathetic tendency often remains dormant for long periods—even months and years. During these periods of abeyance the urine is in every respect perfectly normal, and shews no appreciable defect in the excretion of uric acid.

The Blood in Gout.—The serum of the blood in gout is impregnated with urates; that is to say, a condition which may be termed *uratæmia* prevails. This was first discovered by Sir A. Garrod in 1847, and has since been amply confirmed. Traces of urates exist, often at least, in normal blood, but so minute that they can only be detected by an elaborate process of analysis. In the gouty state, on the contrary, the blood is so surcharged with urates that their presence in the serum can be easily demonstrated by a simple method, devised by Garrod, and called by him the "uric acid thread experiment." This experiment is carried out in the following manner:—A couple of drachms of the serum are placed in a watch-glass and mixed with twelve drops of strong acetic acid. One or two ultimate fibres from a piece of linen fabric are then immersed in the acidulated serum. The watch-glass is then set aside in a warm place for a day or two until the serum is evaporated almost to dryness. The watch-glass is then placed on the stage of the microscope and examined with a magnifying power of fifty or sixty diameters. If the blood contain urates in excess, minute rhombic crystals of uric acid will be found sprinkled upon the submerged threads; if the blood be normal, no such crystals will be discoverable. By the same method Garrod discovered that in gouty persons the serum effused by the application of a blister is similarly charged with urates. In making this observation he found that it was necessary to raise the blister on a portion of the skin not involved in the gouty inflammation, for blister-serum obtained from the surface of an inflamed gouty joint contained no urates; and he inferred therefrom that uric acid was destroyed in the process of gouty inflammation. The same observer states that during the period of convalescence from acute gout there is a marked diminution of urates in the blood. In the long intervals which, in the earlier periods of the disease, often elapse between the attacks, no appreciable amount of urates is

discoverable in the blood; but in inveterate cases the blood, even in the intervals between the exacerbations, was always found surcharged with urates.

Gout is, however, not the only morbid condition in which urates are in excess in the blood. Such excess has been found also in cases of leukaemia, pneumonia, anaemia, and Bright's disease. Some observers have not only had great difficulty in detecting traces of uric acid in the blood normally but they have also failed to detect it in excess in gout, and the suggestion has been made that uric acid is present in the blood in the form of a complex organic compound that does not yield the ordinary tests. Uric acid is known to form a compound with thymic acid which not only does not yield the ordinary reactions for uric acid but is also not precipitable by the reagents that precipitate uric acid. Schmoll considers that uric acid may be present in the blood normally in this form, and that in gout where it is precipitable from the blood it is no longer combined in this manner. According to this view uric acid would not be present necessarily in excess in the blood of the gouty but in an abnormal form.

Diagnosis.—Articular gout is liable to be confounded with rheumatism—acute and chronic; with rheumatoid arthritis; and, more rarely, with gonorrhoeal, pyaemic, or traumatic synovitis.

As a rule the diagnosis of acute articular gout is easy; but exceptional cases of difficulty occur. The gouty character of the inflammation is affirmed by the discovery of uratic concretions in the rim of the ear or elsewhere. Gouty paroxysms are usually recurrent; and the attack is either inexplicable, or is traceable to dietetic errors or mental worry, but very rarely to cold. Rheumatic attacks, on the other hand, are nearly always traceable to some distinct exposure to cold. The family history of the patient often yields important information. Gout runs strongly in families; and if inquiry discloses that the ancestors were subject to gout, that fact, in doubtful cases, is highly significant. Of almost equal significance are the past mode of life and the dietetic habits of the patient, especially as regards the use of alcoholic beverages.

Age and sex have an important bearing on the diagnosis. Acute gout is not often seen before the age of thirty or thirty-five, unless the hereditary tendency be very pronounced. Rheumatic fever, on the contrary, is most common in early life between the ages of fourteen and thirty. The female sex is very markedly more exempt from gout than from rheumatism. The symptoms in gout and rheumatic fever are usually quite distinctive. In gout the fever is less intense, while the local pain is more severe; the larger joints (except the knees) are less generally implicated, and there is an absence of cardiac complications. Severe sweating is not nearly so conspicuous in gout as in rheumatic fever. When the attack is subsiding oedematous pitting is usually observed in gout about the swollen joints, and is followed by desquamation. These signs are not observed after rheumatic inflammation.

It is in cases of chronic and irregular gout that the diagnostic

difficulties are most embarrassing. The convenient term *rheumatic gout* is no longer in favour; and, no doubt, cases so designated are in the majority of instances examples either of pure gout or of pure rheumatoid arthritis. Nevertheless it may be said that in exceptional cases the two conditions are combined, and to such cases the name rheumatic gout may be correctly applied. There is no obvious reason why gouty subjects, especially those of the asthenic type, should be exempt from rheumatic and rheumatoid affections; and cases occur, more commonly among women than men, in which the combination of symptoms and physical signs indicates an overlapping of the two morbid conditions, and in which the designation of rheumatic gout is strictly appropriate.

Prognosis.—The prospect of survivorship in gout depends largely on the presence or absence of complications. Members of gouty families are often long-lived; and if the kidneys remain sound, and the periodical attacks are not too frequent and too protracted, the general level of health is not appreciably lowered. The appearance of albumin in the urine is always an untoward sign in gout, because it too often portends the implication of the kidneys in the gouty process. It is, however, important to be aware that gouty persons may have traces of albumin in the urine for many years, even to old age, without falling into genuine Bright's disease.

Treatment.—The treatment of gout ranges over a wide field—according to the phases of the complaint, the kind and nature of the local manifestations, and the age, temperament, and antecedents of the patient. Attention will be directed first to the treatment of the gouty paroxysm; in the second place the general management of the gouty state will be considered—the means to be adopted to prevent or lessen the frequency and severity of the recurrent attacks, and to relieve the irregular manifestations of the diathesis.

Treatment of the Gouty Paroxysm.—The treatment of acute gout is conducted, in the main, on the same lines as that of other inflammatory ailments. At the outset the bowels are relieved by a suitable purgative: if the fever run high, the patient is confined to bed; the inflamed joints are kept at rest; the diet should consist of milk and farinaceous articles; and diluents should be freely administered. It is necessary, however, to bear in mind the specific character of the inflammation. Gouty patients bear bleeding badly, and neither venesection nor the application of leeches should be recommended. The application of cold to the inflamed joints must be strongly deprecated: such a proceeding involves serious risk of metastasis of the morbid process to the internal organs. The joints should be simply swathed in cotton wadding covered over with oil-silk. This appliance acts as a light poultice, and promotes cutaneous transpiration and a kindly development of the tumefaction, which is usually followed by great relief of pain. Repeated purgation is undesirable; it is of more service to encourage the action of the skin and kidneys than to induce watery intestinal discharges. During the inflammatory period the use of meat should be avoided; there seems no doubt that neglect of

this precaution tends to prolong the attack, and adequate nourishment of a less stimulating character can be supplied by means of milk, bread, farinaceous puddings, and a little fish. Alcoholic beverages, except in special circumstances, should be withheld during the febrile period. In weak or elderly persons, whose systems have been permanently lowered by repeated attacks, the diet should not be unduly meagre; nourishing soups, white meats, and a modicum of alcoholic stimulants should be allowed. In sthenic cases, during the febrile period, the acidity of the urine should be controlled and its volume increased by the systematic exhibition of bicarbonate or citrate of potash. Salines containing soda should, for chemical reasons, be avoided. If a saline aperient be thought desirable, the sulphate of magnesium should be chosen. The occasional use of mercurials for the relief of hepatic congestion is advisable. When the nights are very restless a Dover's powder, or other opiate, may be administered at bed-time.

For the purpose of controlling the gouty inflammation and shortening the attack there is no remedy comparable with colchicum. Ten to twenty-five drops of the wine or tincture may be given two or three times a day, or a grain of the extract may be given at bed-time in a pill with a few grains of Dover's powder. The use of colchicum requires care; some persons are exceptionally sensitive to the action of the drug, and experience a sense of faintness from heart failure unless the dose be very small. In other persons ordinary doses of colchicum induce purging, and the dose must be reduced accordingly. The striking effect of colchicum in reducing gouty inflammation, and in diminishing the pain, is often very remarkable, and is, undoubtedly, of a specific character. Fears have been expressed that, although the immediate action of colchicum be favourable, the more remote effects are not salutary, and that the use of the drug tends to increase the frequency of the recurrence of the paroxysms. These apprehensions do not, however, appear to rest on any solid ground of facts. When colchicum proves ineffective, or is badly borne, iodide and bromide of potassium, salicin, the salicylates, and the salts of lithia may be used.

When convalescence is fairly established the stiffened and swollen joints should be cautiously massaged and gently exercised; the patient should be encouraged to take the air; the dietetic restrictions should be gradually and tentatively removed; nux vomica, mineral acids, or other suitable tonics should be administered; and, finally, change of air to the seaside or to an upland station is to be recommended, as helpful to accelerate the restoration to health.

Treatment of Chronic and Irregular Gout—the General Management of the Gouty State.—The fundamental aim of a rational treatment of chronic gout must always be to diminish the incidence of the diathesis on the constitution. According to the view set forth in this article the essence of the gouty diathesis consists in an enduring tendency to the accumulation of urates in the bodily fluids and to their precipitation as crystals of sodium biurate in the tissues. The means we possess of influencing the factors

which govern the precipitation of sodium biurate in the body may be divided into those which belong to the domain of diet and regimen, and those which consist in the administration of medicinal substances.

Diet and Regimen.—It has been shewn that one of the main factors in determining uratic precipitation is the percentage of urates in the medium. Other things being equal, the larger the proportion of urates present, the earlier and more abundant will be the deposition of the crystalline biurate. Our power of controlling this factor lies almost entirely in the direction of regulation of the diet. Numerous series of experiments have been made on the effect of diverse kinds of food on the production and excretion of uric acid. The point of chief therapeutical interest which has been clearly made out is this: that the ingestion of large quantities of protein matter is attended with an increased production of uric acid, and vice versa. It does not appear clear that protein substances derived from the animal kingdom differ in this respect from those derived from the vegetable kingdom. Nevertheless, inasmuch as the commonly used articles of food of animal origin—such as butcher's meat, poultry, game, fish, eggs, and cheese—are richer in protein stuff than the commonly used articles of vegetable origin—such as bread, oatmeal, rice, potatoes, and garden produce—it is true that a vegetable diet is less productive of uric acid than an animal diet. The most trustworthy experiments indicate that fat, starch, and sugar have not the least direct influence on the production of uric acid; but as the free consumption of these articles naturally operates to restrict the intake of nitrogenous food, their use has indirectly the effect of diminishing the average production of uric acid. There may be, and indeed undoubtedly are, other differences between animal and vegetable articles of food, and between one article and another of the same class, which are highly important. Articles of diet differ considerably among themselves in their digestibility and in their stimulating qualities; but in regard to the point under notice, namely, their direct influence on the production of uric acid, articles of diet must, so far as our present knowledge goes, be classified according to the percentage of albuminoid matters contained in them. Burian and Schur's observations shew that the quantity of uric acid excreted is not influenced by the quantity of protein food ingested, but by the presence or absence of purin bases in such proteids. Meat is harmful not from the high percentage of protein matter but from the presence of hypoxanthine and xanthine. The flesh and glands of all animals contain considerable quantities of these purin bases, whereas milk, cheese, and vegetables contain but little. Cheese is probably not harmful except in so far as some varieties may be indigestible. As a rough guide in the choice of food for the gouty, the subjoined table may prove useful:—

[TABLE

TABLE shewing the Average Percentage of Albuminoid Matters contained in diverse Articles of Food.

Animal Food.	Albuminoid Matter.	Vegetable Food.	Albuminoid Matter.
Butcher's meat	19 per cent	Bread	8 per cent
Fowl	20 ,,	Oatmeal	12 ,,
Game	22 ,,	Rice	6 ,,
Fish	17 ,,	Green peas	6 ,,
Egg	13 ,,	Potatoes	2 ,,
Milk	4 ,,	Carrots and turnips	1 to 2 ,,
Cheese	30 ,,	Green vegetables and salads	1 to 2 ,,
		Fresh fruit (excluding nuts)	0·5 to 1 ,,

In choosing a diet for persons disposed to uratic precipitations, regard must be had of course to the whole condition, and especially to the peculiarities of the individual. Nowhere, perhaps, is it more necessary than in gout to consider the man as well as the ailment, and very often more the man than the ailment; nevertheless the general rule of diet seems pretty clear. Gouty people should be advised to partake cautiously of butcher's meat, fowl, game—and to partake as freely as their digestion will permit of bread, rice, garden vegetables, salads, and fruit. The advantage to be gained from an adjustment of the dietary on these lines may be inconsiderable, or even inappreciable, in cases of inveterate gout; but it may be of critical moment in the slighter cases. A diminution of one or two grains per day in the amount of urates thrown into the circulation may make all the difference between the occurrence or non-recurrence of an arthritic attack.

Alcoholic Beverages.—The highly important part played by certain kinds of alcoholic beverages in the genesis of the gouty constitution, and in fostering a proclivity to uratic precipitation, has been well established. The precise mode in which this adverse influence takes effect is not well understood. The most trustworthy researches indicate that these beverages, in their legitimate use, exercise no appreciable direct influence on the quantity of uric acid produced in the body. Nor is their action proportionate to the percentage of alcohol contained in them. Distilled spirits have but little influence in promoting gout, and whisky and gin less than brandy. On the other hand, the richer wines—port, sherry, Madeira, champagne, Burgundy—and strong ales and stout are highly provocative of gouty manifestations. The most wholesome wines for the gouty are clarets and hocks. Fully fermented, mature, and dry wines are less injurious than sweet and new wines. In gouty persons with a robust habit of body a regimen of total abstinence from alcoholic stimulants is generally the best. Practical experience, however, shews that hard and

fast lines on this matter cannot be maintained. A good many gouty persons, as a matter of fact, fare better with a moderate allowance of alcoholic liquors than without any. With some cases of this class the complete withholding of stimulants fosters a low asthenic type of gouty manifestations, which are more injurious to the general health than frank inflammatory attacks. The personal experience of an observant patient in regard to the point under consideration should always be elicited and carefully weighed by the medical adviser. Such experience not infrequently furnishes valuable indications which may be turned to good account in the general management of the case.

The Use of Common Salt.—It has already been pointed out that all sodium salts act very adversely on the solubility of sodium biurate, and greatly promote its precipitation. It has also been shewn that the topographical distribution of uratic deposits throughout the body bears a striking relation to the percentage of sodium salts contained in the several organs and tissues. In fact, the chemical evidence before adduced seems to point to the deduction, that if we possessed the power of regulating the proportion of sodium salts in the fluids and tissues of the system, we should be able effectively to control the occurrence of uratic precipitations. Our power in this respect, however, is restricted: sodium salts belong to the physiological constants of the blood, and their proportion therein can only be altered through a limited range. These remarks apply especially to the most abundant of them, the sodium chloride. It has been found, in experiments on animals, that when common salt is given in excess with the food, or injected into the veins, the surplus is for the most part quickly removed by the kidneys, and thus increase of its percentage in the blood is but small and transient. And, conversely, when animals are fed with food abnormally poor in salt, there is but a slight falling-off in its proportion in the blood. The blood clings with great tenacity to its proper percentage of sodium chloride; and the experimental evidence indicates that in case of a threatened salt-famine within the economy the blood has the faculty of supplying its necessities by extracting salt from the less vital fluids and tissues: contrariwise, in case of a glut of salt in the blood, the overplus is temporarily passed over into the serous cavities until such time as the kidneys have succeeded in restoring the normal equilibrium. These observations lead to the inference that by lessening the intake of salt with the food we should only abate its proportion in the blood to a slight degree, but should diminish its proportion in the synovial fluids and fibrous tissues considerably. Acting on these opinions, Sir W. Roberts advised gouty patients to restrict their use of sodium chloride as a condiment, and to substitute for it, as far as practicable, the use of potassium chloride, and found that this plan of treatment followed out for years apparently had the effect of preventing or diminishing the recurrence of arthritic attacks, and of removing the various manifestations of irregular gout.

Administration of Medicinal Substances.—The medicinal agents chiefly employed in the treatment of gout, with a view of controlling the tendency

to uratic precipitation, are the alkaline carbonates, the salicylates, the carbonate of lithia, piperazine, and the waters of mineral springs.

Alkalis.—Alkaline substances are largely employed in the treatment of gout, both as pharmaceutical preparations and as components of mineral waters. It is widely believed that the alkaline carbonates administered internally, by increasing the alkalescence of the blood, enhance its solvent power on the material of gouty deposits, and thereby delay or prevent their formation. The experimental evidence before adduced entirely destroys this hypothesis. It has been conclusively proved that alkalescence, as such, has no influence whatever on the solubility of sodium biurate. It has, moreover, been shewn that the addition of an alkaline carbonate to blood-serum impregnated with uric acid produces no appreciable effect on the process of maturation, or on the advent of precipitation of the crystalline biurate in the medium. The use of alkalis in gout has been advocated on another ground. It is held, in a vague sort of way, that there is an undue prevalence of acid in the gouty system, and that the blood is less alkaline than it should be. In some quarters it is even believed that this is the primary vice of the gouty state, and that there exists a so-called "acid dyscrasia" which dominates the whole condition. I have been at some pains to ascertain what foundation there is for this belief, and have found very little of any kind, and none that is really valid. In the numerous examinations of the blood in gouty subjects made by Sir Alfred Garrod, the serum was invariably found to be alkaline, never acid or even neutral. But he remarks that there is often (not always) a marked alteration in the degree of its alkalinity, and that in cases of chronic gout the serum sometimes shews a near approach to neutrality. It is, however, obvious that observations on the alkalinity of the blood have no validity in regard to the point under consideration unless they are made on cases of gout pure and simple. Gout is often complicated not only with pyrexia, but with serious secondary lesions in the kidneys and joints, which lead to a profound cachexia. These secondary lesions bring with them blood changes of their own, which are but remotely connected with the primary disorder, and have no bearing on the etiology of uratic precipitation. Exact quantitative measurements made on the alkalinity of the blood both in health and in disease indicate that a diminished alkalescence of the blood is a common pathological deviation, and that it occurs in conditions which have no relation to gout—namely, in pyrexia, diabetes, carcinoma, acute rheumatism, anaemia, leukaemia, and apparently in every kind of general cachexia. These facts and considerations suffice to shew that, in the present state of our knowledge, the belief in an acid dyscrasia in gout rests on a pure assumption. Lastly, the use of alkalis in gout is advocated on the ground that they facilitate the task of the kidneys in separating uric acid from the blood. The experimental evidence hitherto adduced on this point is so hopelessly contradictory that no conclusion can be safely deduced from it.

Clinical experience on the use of alkalis in gout speaks with a

doubtful voice. Sir W. Roberts used alkalis freely and in sufficient doses to maintain the urine persistently alkaline, yet in these very cases the arthritic attacks recurred with apparently unabated regularity.

Salicylates have been largely tried in the treatment of gout, both in this country and on the Continent. The clinical evidence as to their efficacy is very contradictory. The use of salicylate of soda is open to the same objections as apply against the use of all soda salts. The same objections do not apply to the salicylates of lithia and potash.

Salts of Lithia and Piperazine.—Lithium salts were first introduced into medical practice by Sir A. Garrod, and they are now extensively employed in the treatment of chronic gout. The preparations most commonly employed are the carbonate and citrate, which are given either simply dissolved in water or in the guise of an effervescent water, as "lithia water," containing 5 to 10 grains in the pint. Piperazine is a more recent introduction, and is administered in the same ways and in similar doses. Carbonate of lithia and piperazine have been recommended for the treatment of gout expressly on chemical grounds. Watery solutions of these substances possess a considerable solvent power on free uric acid; and it has been inferred from this fact that their administration internally might exercise a favouring influence on the solubility of sodium biurate in the bodily fluids, and thereby tend to prevent the formation of uratic deposits. This inference does not, however, appear to be justified, because the solvent relations of free uric acid and of sodium biurate are widely different. It was found experimentally that the addition of carbonate of lithia or piperazine, in the proportion of 0·1 per cent and 0·2 per cent, to blood-serum or synovia had not the slightest effect in enhancing the solvent power of these media on sodium biurate, nor the slightest effect in retarding its precipitation from serum and synovia artificially impregnated with uric acid. If these bodies have any beneficial action in gout it is certainly not due, as has been supposed, to their solvent action on the material of gouty concretions. Lysidine, sidonal, urosin, have been credited with the power of increasing the excretion of uric acid. Citarin, the anhydromethylene citrate of sodium, has been recommended at the onset of acute attacks, and has led not only to relief of pain, but also, it is stated, to rapid subsidence of the attack.

Mineral Springs.—The facts set forth in the preceding portion of this article respecting the solvent relations of sodium biurate have an important bearing on the use of mineral springs in the treatment of gout. A considerable number of the springs to which gouty patients resort are strongly impregnated with sodium salts; and it has been conclusively shewn that all the salts of sodium exercise an adverse influence on the solubility of sodium biurate, and hasten its precipitation. It is not, therefore, surprising to learn that not infrequently the first effect of these waters on a gouty patient is either to provoke a downright attack of gout, or to aggravate the symptoms under which he was suffering. This event is now recognised by many physicians practising at these

spas as a thing to be looked for; and experience has taught them the necessity of caution in regard to the quantity of the waters to be taken by newcomers. They comfort themselves and their patients, however, with the assurance that this preliminary storm is a necessary prelude to the calm amendment which is to follow. There is, no doubt, some foundation for this idea. It is no fiction that a gouty man, tormented by symptoms of irregular gout, is relieved by a regular arthritic attack. It may be presumed that this relief arises from the complete, or approximately complete, precipitation into the structures of the joints of the urates floating in his blood and lymph. The urates are thereby almost as effectually removed from the vital fluids as if they were eliminated by the kidneys. Sir W. Roberts was of opinion that gouty persons should either entirely avoid springs which owe their activity to sodium salts, or should use them very sparingly; and that any good effects produced by them must be due to their action on the liver and the intestinal tract.

There are, however, other springs of high and growing repute in the treatment of gout which are not open to these objections. These springs contain no soda, or traces only; and the sum of their mineral constituents does not exceed that which is often present in ordinary potable waters. They contain for their principal ingredient a little carbonate or sulphate of lime; and it is very doubtful whether the whole of this is absorbed into the blood: most of it probably passes out inertly with the faeces. In fact, springs of this class may practically be considered as equivalent to ordinary drinking-water, except that several of them have the advantage of being thermal. Among springs of this class may be mentioned: in our own country, the waters of Buxton, Bath, and Strathpeffer; in Germany, the waters of Gastein, Wildbad, Pfeffers, and the Sauerling spring at Carlsbad; in France, the waters of Aix-les-Bains, Contrexéville, Vittel, and Barèges. Now, there can be no reasonable doubt that the efficacy of these springs has nothing to do with their scanty mineral ingredients, but depends essentially on their watery constituent. They are drunk freely, and on an empty stomach. Their action would be to dilute the blood temporarily, and lower its percentage of urates and sodium salts. This effect would tend to retard or prevent uratic precipitation, and thus give the defective kidneys additional time to overtake their arrears in the task of eliminating uric acid. The muriated sulphurous and sulphurous spas (Schinznach, Uriage, Harrogate) have also been recommended, and probably owe their reputation to their action on the intestinal tract.

It may be asked whether the drinking of water at home would not answer as well as resorting to a mineral spring. The inference from the foregoing observations is that, other things being equal, the beneficial results would be the same. But the "other things" never are "equal." It would scarcely be practicable for a man going about his usual business to drink eight or ten tumblers of water on an empty stomach every day for two or three weeks: at a watering-place the visitor has nothing else to do than to attend to his "cure." Moreover, in getting away from home

the invalid leaves behind him the worries of his daily life, and has the advantage of change of air and scene, a salutary modification of diet, and abundant leisure for outdoor exercise. These collateral influences help to raise the general level of health, and quicken the action of the secretory cells. Besides, the waters of mineral springs are not merely used in the way of drinking; they are also used in the way of baths and various kinds of douches; and the proper hydrotherapeutic treatment is usually supplemented by shampooing, passive motion of the stiffened joints, electric baths, hot packs, and other appliances, all of which are of great service in chronic gout. I do not think, therefore, that gouty patients, if they can afford the time and expense, should forgo the advantages of the time-honoured practice of a visit to a mineral spring. At the same time, a word may be said in favour of a more systematic use of water in the everyday life of the gouty. I have observed that some gouty persons are very sparing in their use of diluents; such persons should be encouraged to be habitually more liberal in this respect. In a few cases it might even be possible to imitate, with plain water, the regular two or three weeks' course at the spa, and to repeat this course twice or thrice a year, as a prophylactic measure.

General Hygienic Management.—Sedentary occupations and idle habits of life are highly injurious to gouty persons, and tend strongly to provoke recurrent arthritic attacks, and to engender the various irregular manifestations of the disease. Gouty patients should be encouraged to lead an active out-door life; they should take exercise systematically in the open air in the way of walking, golfing, horse-riding, or driving; and should, as far as practicable, avoid heavy dinners and late hours. Due foresight should be exercised to evade anxious and worrying engagements, and to guard against undue mental or bodily fatigue. The body should be clothed in light flannel, and the action of the skin promoted by baths and friction. Gouty men are often disposed to be neurotic, and torment themselves with vain ponderings over their symptoms. It is not, therefore, desirable, unless there be some special reasons, for them to relinquish their occupations and lapse into a life of idleness. Some persons adopt the practice of taking a tumbler of hot water, or some hot diluent, on going to bed at night and on rising in the morning. I believe that this practice is a wholesome one for gouty constitutions, and tends to clear the blood of uratic impurities.

Local Treatment of Gouty Affections.—As a rule the local manifestations of chronic gout are most effectively combated by means directed to diminish the general incidence of the gouty state, but sometimes they call for separate treatment. The thickening, stiffening, and aching about gouty joints often persist long after the inflammatory process has passed away. In this condition relief may be given by topical measures. Among the means most in vogue for this purpose are:—Painting the joints with iodine, persevering use of wet compresses, friction with stimulating liniments, shampooing and passive motion, and the application of flying blisters. Hot mineral baths, douches, and galvanic baths

have also been employed in such cases with conspicuous success. Subcutaneous tophi in the neighbourhood of joints sometimes become tense and painful, and interfere with the movements of the adjacent articulations. It has been asserted that the persevering application of pledgets soaked in solutions of carbonate of lithia or carbonate of potash to such enlargements has some effect in reducing their size and in dissolving out the urates contained in them. It is not desirable to puncture or cut these swellings, as such procedures are very apt to leave behind them indolent sores which are very difficult to heal. Sometimes such tophi burst spontaneously, and form indolent ulcers, which occasionally penetrate into the joints, and discharge a purulent sanies mixed with uratic crystals. The most effective way of dealing with these ulcers is to immerse the part for some hours daily in a large can of warm water, with a view of dissolving out the offending urates. A similar mode of treatment may afford great relief in cases with uratic deposits encrusting the heels or palms and thereby causing pain and hindering locomotion.

It is not always desirable to interfere too actively with gouty affections of the skin; they often appear to operate as a sort of safety-valve to the gouty system; but sometimes the itching and irritation caused by them become wellnigh intolerable, and seriously interfere with sleep and the general well-being of the patient. In such cases constitutional measures must be eked out by local means. Solution of borax with glycerin, boracic acid in vaseline, lead lotion, zinc ointment, bismuth and starch powder applied to patches of gouty psoriasis, eczema, or prurigo often afford great relief. In the case of dry eruptions, associated with much itching, the solid paraffin is a valuable resource. A piece of hard paraffin of the size of the thumb is pared down to a smooth surface with a penknife, and the itching surface is rubbed over morning and evening with this, so as to leave a delicate coating of paraffin on the skin. It is a perfectly cleanly and wholly invisible application; and probably acts by protecting the cutaneous surface against the contact of the atmosphere.

WILLIAM ROBERTS, 1897.
J. ROSE BRADFORD, 1907.

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J. R. B.

DIABETES MELLITUS

By Robert Saundby, M.D., M.Sc., LL.D., F.R.C.P.

Synonyms.—*Diabetes; Saccharine diabetes; Glycosuria; Mellituria; Diarrhoea urinosa; Polyuria.*

Definition.—A constitutional disease characterised by the persistent passage of glucose in the urine.

History.—In the papyrus Ebers, which is a copy of an Egyptian medical compilation already old in the time of Moses, there is mention of polyuria, and it is hard to conceive that such a marked departure from health could at any time have escaped observation; yet no notice of it is to be found in Greek writings earlier than those of Aretaeus of Cappadocia (*circa* A.D. 150), who is supposed to have been a contemporary of Galen, and was probably also a Roman physician. His description, like that of the unknown Egyptian priest, or that of Galen, who wrote at length upon the disease, lays stress only on the polyuria and thirst. The important point that the urine contains sugar appears to have escaped the notice of all early European writers; nevertheless, if we may trust some passages of the Ayur Veda (*circa* A.D. 500) it was known at that time to the Hindus. In a Cingalese writing of the fifteenth century diabetes is undoubtedly referred to as "madu mehe," or *honey urine*, so that in this respect European knowledge lagged sadly behind; for it was not until 1679 that our countryman Willis mentioned that the urine of diabetes has a sweet taste. A century later Dobson of Liverpool demonstrated the presence of sugar, which discovery so impressed Cullen, that he would hardly allow that this was not true of all cases of diabetes; and not until the last century has the distinction been firmly established which we recognise in the names *diabetes insipidus* and *diabetes mellitus*.

Etiology.—The mean annual mortality from diabetes mellitus in Europe does not exceed 5 per 100,000 of persons living. But within this area considerable variations may be noted: for example, in Paris [1] the figure is as high as 14, in Malta 13, in Copenhagen 7, in London 7, in Vienna 4, and in Naples 3. In the whole of England it is 7, in Ireland 3, in Scotland 2, in Norway 2, in Prussia 2, and in Italy 1·5. Diabetes is said to be extremely common among the educated and commercial classes of natives in India and Ceylon, while it is unknown among the Chinese and Japanese and the negroes of Africa. This immunity of the negro appears to persist, partially, at least, in America; for Tyson of Philadelphia has stated that he has never met with a case in the negro, although he has modified the inference to be drawn from his personal

[1] *Vide* table at foot of page 168.

experience by informing me that he has since heard of two examples in the practice of a coloured physician.

American authors formerly asserted that their national statistics proved the annual rate of mortality from diabetes for all races to be much lower in the States than in Europe; but, as anticipated, better statistics have shewn that in 1902 the rate per 100,000 living in the city of New York was 14·40 (Herman M. Biggs), while in Philadelphia in 1900 the rate per 10,000 deaths was 43·4, figures which shew New York to be as bad as Paris, and Philadelphia little better than London.

In France the disease is said to prevail extensively in Normandy; in Italy it is apparently more common in Tuscany than in other parts of the kingdom.

Scudamore was certainly mistaken in the assertion that the Scottish people are specially liable to diabetes; and his unsupported statement, completely contradicted as it is by the Registrar-General's Reports, would not call for any protest had it not been quoted and accepted by Hilton Fagge, who founded upon this supposed prevalence, taken together with the known rarity of gout in Scotland, the wrong conclusion that gout and diabetes are in nowise related.

All observers are agreed that Jews are specially liable to become diabetic. There is some reason to believe that modern city life is in itself a cause of diabetes. In his admirable statistics for Paris (see table, p. 169), Bertillon has demonstrated that the mortality from diabetes is higher in all the wealthier *arrondissements*; while in India and Ceylon it is among

Deaths from Diabetes in Paris (Bertillon).

Year.	Number of Deaths from Diabetes.	Proportion per 100,000 Population.
1892	295	12
1893	349	14
1894	294	12
1895	359	15
1896	378	15
1897	389	14
1898	387	15
1899	373	14
1900	413	16
1901	380	14
1902	376	14

England—Wales.

Year.	Deaths from Diabetes.	Total Deaths.	Proportion per 10,000 Deaths.	Proportion per 100,000 Living.
1892	2011	559,684	35·9	7
1902	2769	535,538	51·7	8·5
		London.		
1892	292	86,833	33·9	7
1902	356	77,829	46·0	7·8

the educated and commercial and not among the labouring classes that the numerous cases of diabetes occur. A person belonging to the richer classes in towns usually eats too much; spends a great part of his life indoors; takes too little bodily exercise, and overtaxes his nervous system in the pursuit of knowledge, business, or pleasure. Although there are many exceptions to such a description, it is a perfectly accurate account of large classes, and especially of the well-to-do Jew, who raises himself easily by his superior mental ability to a comfortable social position, and notoriously avoids all kinds of bodily exercises.

The excessive use of sugar as food is very generally believed to be a cause of this disease, although it is not easy to see how it acts. The fondness of Jews for sweet things has been suggested as one explanation of the frequency with which they become diabetic; and local practitioners in Persia, India, and Ceylon are accustomed, rightly or wrongly, to attribute the disease to abuse of sweetmeats and sweet sherbets. Even the exclusive employment of vegetable food has been blamed for the same reason; and Charcot stated that temporary glycosuria is often observed among the novices at La Trappe. But in Mauritius and British Guiana—countries in which the people are chiefly employed in the manufacture of sugar, in Africa where the sugar-cane and other sweet fruits and vegetables form a large part of the diet of the natives, and in Ireland where the food of the peasantry is mainly vegetable, the disease is relatively rare. As little valuable are the opinions that beer-drinking and cider-drinking are efficient causes: the deaths from diabetes in the cider-drinking counties of England are below the average, while the kingdom of Prussia, which probably stands at the head of the beer-consuming countries of Europe, has but a low diabetic mortality.

TABLE shewing the Average Annual Mortality from Diabetes per 100,000 of Population for each *Arrondissement* of Paris during three quinquennial periods.

Name.	1865-69.	1881-85.	1886-90.	Remarks.
Louvre	3	5	11	Very well-to-do.
Bourse	2	7	10	,, ,,
Temple	2	12	16	Well-to-do.
Hôtel-de-Ville	3	8	13	,,
Panthéon	2	6	10	,,
Luxembourg	3	11	15	Rich.
Palais-Bourbon	4	9	14	,,
Elysee	4	12	16	Very rich.
Opéra	5	13	20	Rich.
St. Laurent	4	10	12	Very well-to-do.
Popincourt	2	6	11	Poor.
Reuilly	2	8	11	,,
Gobelins	1	8	9	Very poor.
Observatoire	2	7	9	Poor.
Vaugirard	1	6	14	,,
Passy	2	12	18	Rich.
Batignolles	3	8	14	Well-to-do.
Montmartre	1	7	9	Very poor.
Buttes-Chaumont	1	4	7	,,
Ménilmontant	2	5	8	,,
All Paris	4	8	13	
Males	3	10	15	
Females	2	6	10	

No *age* is exempt, but diabetes occurs most commonly in persons at the fifth decade of life, being met with more rarely at its two extremes. Cases have been observed in infants a few weeks old, and in aged persons over eighty. The greatest mortality is between the ages of fifty-five and seventy-five—that is some years later than the period at which it most frequently supervenes, for elderly diabetics are able to sustain the disease for many years.

The different incidence of the disease in the two *sexes* is well marked in tables [1] which include deaths at all ages, being in the proportion of 3 males to 2 females. This difference is least marked in young persons between the ages of 10 to 35, and most marked between 35 and 75, due to the greater prevalence of the abuse of alcohol among males.

Heredity is an influence too well attested in certain instances to be doubted; thus, in one example diabetes occurred in eight members of one family extending over three generations.

It is also not uncommon to meet with a history of *insanity*, *pulmonary tuberculosis*, or *gout* among the relatives of diabetics.

Obesity is supposed by many persons to favour the occurrence of diabetes: undoubtedly the coincidence is common enough, but the opinion of Kisch, that it directly arises from the muscles becoming so infiltrated with fat that they are thereby unable to do their share in the destruction of sugar, is open to doubt. It is more probable that obesity is the outcome of similar general conditions. Where the supply of carbohydrates is in excess of the demands of the tissues or of their consuming capacity, it is stored as fat; and if we regard obesity as an indication of such an excess, it is not difficult to suppose a further stage at which, the limits of fat-storage being reached, the surplus sugar circulating in the blood passes out of the body by the kidneys, and glycosuria, transient or permanent, ensues. The excessive supply of carbohydrates, whether in the form of sugar, vegetable food, or beer, does no harm in the absence of the essential causes of diabetes; the excess is stored up as fat, or may even appear in the urine as a transient glycosuria. But when an excess of glucose, greatly beyond the power of the organism to utilise, is constantly poured into the circulation, we have the condition set up that we call diabetes; this must appear all the more rapidly where the carbohydrate supply is already more than can be consumed.

[1] England and Wales.

All Ages.	Under 1 year.	1 year.	2 years.	3 years.	4 years.	Total under 5 years.	5 years.	10 years.	15 years.	20 years.	25 years.	35 years.	45 years.	55 years.	65 years.	75 years.	85 and upwards.
Males, 1142	3	4	2	1	1	11	3	30	44	47	116	130	192	262	233	67	7
Females, 869	..	..	..	1	3	4	7	28	34	48	104	84	125	198	177	57	3
Total, 2011	3	4	2	2	4	15	10	58	78	95	220	214	317	460	410	124	10

See also Vol. I. p. 74.

The exact relation of *gout* to diabetes is an open question. No one who has an adequate clinical knowledge of the two diseases doubts the frequency of their association ; both are due to super-alimentation. It is possible that an acute attack of gout may be the direct cause of the onset of diabetes ; but it is not clear that the gouty diathesis in itself disposes to diabetes except in so far as it is evidence of the pathological results of modern civilisation in certain individuals. There are many gouty families and more gouty individuals in whom the diathesis has its most typical expression without resulting in the production of diabetes.

The list of the *immediate causes* of diabetes is a very long one, but in most instances the relationship reposes on too well established a basis of clinical evidence to be rejected.

In the first place comes the long catalogue of injuries, such as railway accidents, severe contusions of the head, spine, or abdomen, violent muscular strains, and lightning-stroke, which are so frequently followed after a longer or shorter interval by diabetes. In all these cases the disease is probably produced by some derangement of the innervation of the liver. Closely allied, therefore, to these are the rarer examples in which the disease is actually associated with some coarse lesion of the medulla ; as, for example, where this structure is implicated in a new growth, or invaded by sclerosis (tabes dorsalis, insular sclerosis) ; or where it occurs in the course of insanity, epilepsy, or Graves' disease. Possibly we ought to group under nervous influences those cases which have been attributed to climacteric disturbance, excessive sexual indulgence, and the action of certain poisons ; for example, alcohol, chloroform (Fort), bromide of potassium (Weber).

In the second place, but in quite a different category, come the cases which follow the various acute infectious diseases, among which numerous well-attested examples have been recorded as sequels of enteric fever, diphtheria, tonsillitis (?), influenza, rheumatic fever, malaria, and syphilis. Insufficient attention has been paid to the possibility that diabetes has in some cases a microbic origin. Charrin and Carnot have proved that it is possible to render a dog diabetic by injecting infective fluids (*Bacillus coli*, *Bacillus pyocyaneus*, and streptococci) into the pancreatic duct ; and it is probable that the acute infective diseases diminish the resistance of the pancreas to the invasion of those organisms which occur so abundantly in the intestine. There is no evidence at present of any specific microbic cause for diabetes ; but in connexion with this aspect of the subject I may refer to the many examples of diabetes occurring in husband and wife, which have led some writers to speculate on the possibility of its transmission by contagion (Dréfous, Gaucher, Labbé, Letulle, Schmitz, Rendu).

We may group with the above those cases which have been recorded in connexion with pancreatic cancer, cysts, and calculi.

In a third class must be placed the cases which have their apparent starting-point in pregnancy and parturition, as their explanation remains obscure.

Beside the above, are the cases associated with abscess of the liver, malignant disease in the abdomen not invading the pancreas, exposure to severe cold, and the ingestion of cold food (ices) or iced drinks.

Pathogenesis.—Diabetes has been already defined as persistent glycosuria. By this we mean the constant presence of a glucose in the urine in quantity sufficient for discovery by ordinary tests. The glucoses, or hexoses, are ketones or aldehydes derived from hexatomic alcohols; dextrose (or d-glucose, an alcohol-aldehyde) is habitually, and laevulose (or d-fructose, an alcohol-ketone) exceptionally, to be found in the urine of diabetics. The sucroses or disaccharides ($C_{12}H_{22}O_{11}$) are formed by loss of H_2O from two hexose molecules; thus cane-sugar is built up from d-glucose and d-fructose, maltose from two molecules of d-glucose, and lactose—occasionally met with in the urine of nursing women—from d-glucose and galactose. Starch, dextrin, and glycogen are polysaccharides $(C_6H_{10}O_5)_n$, formed by the union and dehydration of several hexose molecules. Under the influence of ferments or the action of dilute acids this synthesis is reversed, and the complex molecule of starch is split up and hydrated to form dextrin, maltose, and d-glucose, as in ordinary digestion.

Traces of dextrose are met with constantly in normal blood (0·5 per mille), and corresponding traces can be detected in the urine by concentrating a large bulk; but it is not with these small quantities that we are concerned in diabetes: in this disease the blood contains excess of sugar (as much as 0·4 per cent) and the urine is loaded with it.

The first light thrown upon the pathology of glycosuria was the discovery by Bernard (1849) that the liver after death contains sugar; and his inference that the liver is a sugar-forming gland has withstood all the criticism to which it has been subjected. Bernard's researches did not stop here; he found that the liver stored carbohydrates in the form of glycogen, and he believed that this substance was converted into sugar under the influence of a diastatic ferment contained in the blood: moreover, by means of puncture of the floor of the fourth ventricle he succeeded in determining such an excessive discharge of sugar that the excess passed into the urine. According to Bernard, carbohydrates are absorbed from the intestine as sugar, and are carried by the portal vein to the liver, where they are stored as glycogen. This stored material is given out gradually as it is required to meet the needs of the economy, being converted into glucose and carried by the hepatic vein into the general circulation.

Bernard's views have been opposed with great ability and pertinacity by Dr. Pavy, but, in spite of much effective criticism of his methods, the result has been to leave his conclusions substantially intact. Bernard's doctrines have found followers able to demonstrate the truth of his statements by more exact means; and at the present time the only important modification towards which opinion seems to tend is the rejection of the agency of a glycogenic ferment in the liver in favour of the conception of glycogenesis as a direct and normal function of the liver-cells.

Dr. Pavy thinks that glycogen may be converted directly into fat;

and there is good ground for his inquiry into the fate of the great excess of glycogen found in the livers of vegetable-feeders, for the blood of these animals contains no more sugar than does that of flesh-eaters, although their store of glycogen is so much greater. Dr. Pavy has made an interesting observation on the presence of fat in the intestinal villi of a long-starved rabbit, which suggests that starch may be converted into fat by the epithelium of the intestinal villi; and he thinks that it shares this function with the liver-cells.

That carbohydrates are not the only possible sources of sugar is proved by the presence of glucose in the blood of animals fed on lean meat only; and our clinical experience of diabetes proves but too frequently how large a quantity of sugar may be excreted by patients whose diet contains very little carbohydrate. Seegen believes that sugar is formed from peptone, and on insufficient grounds denies that glycogen is its principal source; and Dr. Pavy has shewn that sugar may be artificially produced from protein matter. It is certain that sugar can be formed from the protein of the food and from the tissues; and the question whether it is derived from the protein molecule by splitting off, or whether the molecule is first completely broken down and then built up again as carbohydrate and urea, is one which need not detain us; the latter view has, perhaps, the greater weight of authority. The seat of this process, or the agent which determines it in the body, is not definitely known; but it is assumed that it takes place in the liver, and that the liver-cells effect the conversion. But what becomes of the sugar poured into the circulation? In the course of some lectures delivered in May 1905, Dr. Pavy developed the hypothesis that sugar exists in the blood in a glucosidal form and in this way is transported from the intestine to the tissues. Locked up in a large protein molecule the carbohydrate is safe from the danger of being discharged with the urine; these molecules are in his opinion carried by the lymphocytes which appear in the blood in such numbers during the familiar digestion-leucocytosis.

Under the influence of Liebig's teaching it used to be believed that glucose was burnt off in the lungs and excreted as water and CO_2, the process subserving the maintenance of the body-temperature. But of late years the seat of this destructive process has been shifted to the muscles, which are said to store up glucose in the form of glycogen, to be utilised when in action. This view is supported by the disappearance of glycogen from muscles which have been tetanised by strychnine poisoning; and by the destruction of sugar in the tissues proved to take place by comparison of the amount contained in arterial and venous blood respectively.

While Claude Bernard and those who have followed him in the same line of research have done much as pioneers, it cannot be said that they have succeeded in indicating the direction in which the solution of the problem of the pathogenesis of diabetes is to be found. The most careful research has failed to establish that structural disease in the floor of the fourth ventricle or any part of the nervous system or in the liver is even

a common occurrence in diabetes. On the contrary, in the early stages of the disease these organs appear perfectly healthy; so that if they be primarily concerned, diabetes can only be regarded as a *functional disease of the nervous system.*

Besides the "diabetic puncture" the following experimental lesions of the nervous system are said to be followed by glycosuria :—

(i.) Injury to the vermiform process of the cerebellum: (ii.) Section of the spinal cord at various levels: (iii.) Section of the anterior cervical nerve-roots: (iv.) Section of the posterior cervical nerve-roots: (v.) Artificial neuritis of the first pair of dorsal nerves: (vi.) Destruction of the superior and inferior cervical sympathetic ganglia, of the first thoracic, and of the abdominal ganglia: (vii.) Section of or ligaturing the splanchnic nerves: (viii.) Irritation of the right vagus: (ix.) Section and stimulation of the central end of an ordinary sensorimotor nerve such as the sciatic.

Under the influence of these various lesions the glycogenic function of the liver is enormously increased, the quantity of sugar poured into the circulation far exceeds the consuming and storing powers of the tissues, and glycosuria is the inevitable consequence. But the effect of these lesions is, as a rule, transitory, resembling in this respect the majority of cases of traumatic glycosuria in man.

It is probable that some poisons produce glycosuria in the same way, while others are merely converted into reducing substances (for example, glycuronic acid), which are excreted in the urine and are there mistaken for sugar.

In 1877 Lancereaux drew the attention of the French Academy of Medicine to the changes in the *pancreas* which, he believed, were constantly present in severe cases of diabetes; and within recent years the importance of this communication has been demonstrated by the experimental production of diabetes in dogs by extirpation of this gland. Von Mering and Minkowski have found that after this operation the animals suffer from glycosuria, polyuria, great thirst, hunger, and rapid emaciation; in fact, all the classical symptoms of diabetes. On the other hand, mere obstruction of the pancreatic duct—by ligature or by injection with paraffin (Hédon) or asphaltum (Thiroloix)—is not followed by any of these symptoms; nor do they occur if a small part of the gland be left, even although the duct be removed. These statements have been subjected to most severe criticism, and the experiments have been repeated in numberless instances, with the result that their accuracy has been established. It was at first suggested that the operation injured the solar plexus and so set up nervous diabetes; but apart from the statement of the operators, that no such injury had been inflicted, the absence of the symptoms when a part of the gland was left contradicted this view. But the most convincing proof that such an injury cannot be the explanation is afforded by the experiment of grafting a portion of the extirpated pancreas outside the abdominal cavity in the muscles of the external walls; this operation has been successfully performed by

several independent investigators (Minkowski, Hédon, Thiroloix) with the result of preventing the occurrence of diabetes just as if the part of the gland had been left in place.

Inspection of the bodies of diabetic patients has proved that alterations of the pancreas are very commonly present; their nature and frequency will be considered on p. 183. It is enough now to know that they exist, and that experiment and morbid anatomy are not here so strikingly at variance as in other hypotheses of diabetes.

The means by which the presence or absence of the pancreas influences the production of glycosuria needs further explanation. Lépine has endeavoured to find a solution by the supposition that the pancreas secretes a sugar-destroying ferment, which passes directly into the chyle and blood. He asserts that these fluids in the normal state possess such an action to a powerful extent, and that this action disappears when the pancreas has been extirpated; but Lépine's statements are denied by Arthus, Gaglio, and Sansoni: moreover, no one has yet succeeded in obtaining such a ferment from the pancreas, and pancreatic extracts, however administered, have failed to control glycosuria. The pancreatic hypothesis of diabetes is undoubtedly a great advance towards a satisfactory pathology of the disease; but as in certain cases the gland appears to be quite normal, it does not cover the whole ground, and it leaves room for the old view of its nervous origin, at least in respect of a certain proportion of cases. Kaufmann has suggested a modification of Lépine's hypothesis, namely, that the secretion controls the sugar-forming function of the liver; he believes he has proved by analysis of arterial and venous blood in healthy and diabetic animals that the tissues of the latter consume quite the normal proportion of sugar; it was formerly believed that the gaseous exchanges of diabetes gave evidence of diminished oxidation, but the results of later experiments contradict this opinion (Leo, Weintraud and Laves); he therefore regards the hypothesis of insufficient destruction of sugar as untenable. Von Noorden, on the other hand, denies the extra production of sugar, and finds the explanation of diabetes in diminished destruction in the tissues, loss of power to store carbohydrate as fat, and incompetency of the glycogen reservoirs (liver, muscles). He assumes that every molecule of albumin breaks up into carbohydrate and urea, and that for every gramme of urea 2·8 grammes of carbohydrate have been formed in the body; hence he concludes that it is impossible to shew an excretion of sugar greater than may be derived in this fashion.

He disregards the various authors (Voit, Leo, and others) who have shewn that there is no decrease of CO_2 production in diabetes, and attaches great weight to the single observation of Weintraud and Laves, that the addition of small quantities of carbohydrate to the dietary of diabetics raises their respiratory quotient (the proportion of CO_2 to O_2) much less than in health. Finally, he discredits the results of those observers who have found that the sugar is no greater in arterial than in venous blood (Pavy, Beck and Hoffmann, Abeles, Seegen); and although

Chauveau and Kaufmann find that the arterial blood always contains more sugar, this does not help von Noorden, as the relation remains exactly the same in diabetic as in healthy animals.

Where the evidence is so conflicting, and the weight of authority so nicely balanced, we may well hesitate before ranging ourselves on one side of this controversy or on the other. Dr. Pavy believes the intestinal villi share with the liver the power to convert carbohydrates into fat, and he thinks this power is lost in diabetes; but the evidence of this power rests upon his own observation, hitherto unsupported, of the amount of fat demonstrable in the villi of a rabbit after a full meal of oats. Prof. Noel Paton thinks the oats contain enough oil to account for these appearances without assuming for them any less direct explanation. That the carbohydrates are converted into fat (Pavy) or actually consumed (Lépine) before they reach the liver, has not been proved; and either view is as difficult to accept as the ordinary one that all the carbohydrates are first stored in the liver, to be doled out thence in constant, regular measure to supply the needs of the body.

If the liver possess in health not only the power of sugar-formation, but some mechanism by which it regulates the amount passed into the circulation by the current demands of the organism, and thus, whether the store of glycogen be great or small, keeps the percentage of sugar in the blood at a constant point, we have only to assume a disorder of this regulating function to explain the occurrence of diabetes.

In diabetes the liver continues to manufacture sugar constantly, utilising for the purpose its accumulated stores of glycogen, which it soon exhausts, and afterwards the carbohydrates and albumin of the food; apparently falling back upon the albumin when the former are deficient. This would constitute what is called over-production, although the amount may not exceed the value of the food ingested; but in health the quantity of food would probably be less, and the resulting glycogen would be stored up, no more than the required amount of sugar being manufactured to maintain the normal blood percentage.

The formation of sugar in the body takes place in two ways. In the first or more obvious way which has been long known, it is formed by the action of the diastatic and inverting ferments of the saliva and the pancreatic juice upon the starchy and saccharine elements of food. It is generally believed that the resulting sugar is identical with maltose ($C_{12}H_{22}O_{11}$), but by the time this reaches the portal vein it has become glucose ($C_6H_{12}O_6$). Glucose, as is well known, undergoes, in part at least, a further change, and is stored in the liver-cells as glycogen ($6C_6H_{10}O_5 + H_2O$). It has been shewn that the glycogen in the liver is increased by adding starchy or saccharine articles to the diet, and diminished by feeding upon purely albuminous and fatty food, as well as by cold and by violent muscular exercise, while the deprivation of all food causes it to disappear completely. The liver is not the only organ capable of forming glycogen. This power is possessed by muscle, which can form it directly from sugar, as can be proved by injecting syrup into

the circulation; in foetal life glycogen is found stored up in all the tissues of the body, and in adult life, especially in diabetic persons, it may be found in the muscles, kidneys, and elsewhere. Our knowledge of the further stages of sugar metabolism is somewhat conjectural. It is probable that during digestion a certain amount of sugar passes direct into the circulation and is consumed by the tissues, for it has been observed that the livers of rabbits freed from glycogen by starvation shew no evidence of its presence until four hours after a starchy meal, whereas in diabetes increase of sugar in the urine can be recognised in from one to one and a half hours after the ingestion of carbohydrate food. It is probable that the stored glycogen is converted into sugar and passed into the circulation from time to time in very small quantities as the tissues demand it, such calls upon the reserve being occasioned by hunger, exercise, and exposure to cold.

The second way in which sugar is formed has only been made out during recent years by the researches of Kühne, Heynsius, Pavy, and others, by whom it has been shewn that the albumin in animal food, in the course of decomposition into urea, water, and CO_2, splits off a carbohydrate molecule, so that for every grain of urea excreted in the urine we may reckon that at least two grains of sugar have been formed in the body. The sugar thus formed is usually consumed in the body, and it is believed that this consumption is the work of the tissues themselves, being an essential part of their vital activity. With these data it is comparatively easy to estimate the amount of sugar consumed in the tissues in health, for we have only to calculate the daily quantity supplied as carbohydrates and add to this that formed by the decomposition of albumin in order to determine it. Bouchard has shewn that the consumption of sugar varies with age and body-weight, so that young and slender persons consume a larger proportion than those who are older and fatter. Thus, a youth aged 17, weighing 101 lbs., consumed 54 grs. per lb. of body-weight; while a man aged 59, weighing 170 lbs., consumed only 18 grs. per lb. This difference throws a good deal of light upon the well-known predisposition of elderly stout people to glycosuria.

As may be supposed, the capacity of the tissues to consume sugar is not unlimited. It has been demonstrated that it is possible to produce glycosuria artificially by giving large quantities of glucose in solution by the mouth, and it is reasonable to suppose that otherwise healthy persons whose sugar consumption from age and obesity stands low, may easily overstep these limits and excrete a certain amount of sugar which is only the surplus their tissues have been unable to consume. This condition is spoken of as "alimentary glycosuria."

If the whole of the stored-up glycosuria were suddenly discharged into the circulation we should have a condition of temporary hyperglycaemia in which the tissues would be incapable of consuming the whole of the sugar, and some of it would appear in the urine. It has been shewn by numerous experiments, of which the best-known is

Claude Bernard's puncture of the floor of the fourth ventricle, that various lesions of the nervous system cause such a sudden discharge of the hepatic glycogen, which is followed by glycosuria. This was for a long time accepted as an explanation of diabetes, as it was apparently confirmed by the clinical observation that diabetes might occasionally be caused by tumours and other lesions in the neighbourhood of the fourth ventricle, and less uncommonly by falls and blows on the head. It was, however, pointed out that glycosuria due to this source should be easily controlled by withholding carbohydrate food, as the stored glycogen would be very rapidly dissipated. The discovery of the origin of sugar from albuminous food afforded a partial answer to this objection, but the excretion of even a small quantity of sugar by diabetic patients on strict diet cannot be explained unless we assume that the normal consumption of sugar by the tissues has undergone very great reduction. Bouchard investigated this point and shewed that the consumption of sugar by diabetics is always considerably reduced, the reduction ranging from one-half to as little as one-twentieth of the normal amount. We have now, therefore, what appears to be an adequate hypothesis of diabetes, namely, failure of the glycogen reservoir in the liver and reduction of the power of the tissues to consume sugar.

It only remains to shew first what are the conditions in which the liver is no longer able to reserve and store up glycogen; and secondly, what is the cause of the reduction of the sugar-consuming power of the tissues. These conditions are: (1) various functional and organic diseases of the nervous system, of which neurasthenia, whether originating from traumatic shock or from other exhausting agents such as prolonged illness, excessive work, mental strain, strong emotions, must be placed in the front rank; organic diseases of the nervous system are a much more rare cause of diabetes, and consist chiefly of tumours situated in or about the floor of the fourth ventricle or upon the vagi nerves; (2) pancreatic diseases, including fibrosis, fatty metamorphosis, calculous atrophy, cancerous infiltration, and necrosis from acute inflammation; (3) injuries and diseases of the liver itself, under which may be included those cases which have followed blows over the hepatic region, abscess of the liver, and cirrhosis; (4) poisons, of which alcohol is the chief.

A cause of the reduction of the sugar-consuming capacity of the tissues seems to be the hyperglycaemia itself. When the tissues have for some time received an excessive supply of sugar they appear to lose in part their normal consuming capacity, and conversely, after the excess of sugar has been removed by appropriate treatment they may regain this power to a greater or smaller extent.

We may therefore anticipate what is undoubtedly true, that glycosuria occurs in widely differing clinical conditions, but it is only after persistent hyperglycaemia has led to a considerable reduction of the sugar-consuming capacity of the tissues that diabetes sets in. The distinction between diabetes and non-diabetic glycosuria is the superaddition to the glycosuria of the symptoms of thirst, polyuria, and failure

of nutrition. The simplest form of non-diabetic glycosuria is what is called alimentary glycosuria, in which the sugar excreted represents the unconsumed surplus of carbohydrate food.

As has already been remarked, the sugar-consuming power of the tissues varies greatly with age and body-weight. People who have accumulated a thick layer of adipose tissue possess a diminished degree of it, and this defect becomes more marked as they get older. It is therefore easy to explain the glycosuria of fat elderly people, and every practitioner knows how commonly it is seen in such persons. In fact in them a little glycosuria from time to time is normal or quasi-normal, so that we should not be unduly alarmed at its presence, or find any difficulty in explaining it to our patients. No doubt the presence of sugar in the urine indicates how readily hyperglycaemia may be induced by excess, and should such an individual persist in a diet rich in carbohydrates a further reduction of sugar-consuming power may occur with resulting mild diabetes, which, however, is easily controlled, and should not give real cause for anxiety. If temporary glycosuria from excess of sugar occur in early life, when the normal rate of sugar-consumption is high, it reveals a want of power to store and consume sugar which should make us uneasy as to the patient's future, but our clinical experience of these cases is too recent to afford us the data for expressing a positive opinion upon their chance of developing diabetes. In both cases the cause of the glycosuria is simply the want of proportion between the supply of carbohydrate food and the sugar-consuming power of the tissues, but in the other forms of non-diabetic glycosuria there is some pathological agency which affects the glycogen-storing function of the liver either through the nervous system, the stomach, or the pancreas, or by directly attacking the liver itself.

The very interesting, large, and important group of cases in which abuse of alcohol is the real cause of the glycosuria does not deserve to be classed apart for any reason other than its etiology. Alcohol probably acts directly upon the liver, and the glycosuria is the result of the functional derangement caused by it. In many instances the liver has been enlarged, but where no such physical change can be noted it is reasonable to infer that there is functional disturbance. From a pathological point of view the gastric, hepatic, and alcoholic cases may be classed under one head, and attributed to the disturbance of the liver caused by alcohol or other irritating ingesta, or by the products of fermentative or putrefactive change in the contents of the stomach or intestine.

The glycosuria of gout is usually the alimentary glycosuria of elderly fat persons, and I am not sure that the condition ever demands any other explanation. That which follows falls and blows is due to the sudden discharge of the accumulated liver-glycogen. Where neurasthenia follows, the glycogen-storing function of the liver may be regarded as permanently depressed; too much sugar flows habitually into the circulation, and hyperglycaemia with symptoms of diabetes may be set up. The glycosuria caused by boils and carbuncles, ulcers, gangrenous sores, and

many infectious diseases, especially influenza, seems to depend upon toxins of microbial origin. As a rule this glycosuria lasts only so long as the infective process, and disappears when the toxaemia comes to an end. We do not know the channel through which these poisons act, but it is quite conceivable that they attack the liver directly; it is, however, equally possible that the nervous system or the pancreas suffers primarily. Whichever it may be, we are justified by the transitory character of the glycosuria in supposing that the poisons usually produce functional effects only, but as there are cases of diabetes which have originated from attacks of infectious diseases, we must also allow that occasionally they possess the power of producing permanent mischief. As the most common lesion capable of producing permanent glycosuria is wasting of the pancreas, and as there is no satisfactory explanation of its occurrence, we may be permitted to believe that the products of infection at certain times favour the development of inflammation of the pancreas, just as they may give rise to analogous inflammations of the kidneys or liver. We know so little about the true pathology of malignant disease that it is useless to speculate upon the cause of the glycosuria which sometimes occurs in connexion with it, particularly in cases of abdominal cancer. Lastly, senile glycosuria is explained by the greatly reduced capacity of the aged body to consume sugar.

The glycosuria which follows the administration of phloridzin ($C_{21}H_{24}O_{10} + H_2O$), a glucoside obtained from the bark of apple, pear, cherry, and plum trees, is said by Minkowski to be genuine, and not due to the presence of its decomposition product, phlorosin, which has the formula of sugar, reduces cupric oxide, and undergoes fermentation; as the administration of one gramme of phloridzin was followed by the excretion of 97 grammes of glucose, this must be admitted. It is believed that the drug acts by altering the renal epithelium so that it no longer holds back sugar, and this view, as von Noorden points out, reminds us of the old hypothesis of the renal origin of diabetes, and suggests that it possibly plays some part in the pathogenesis of certain forms of the disease.

Cantani's hypothesis, that the sugar of diabetes is a paraglucose incapable of subserving the needs of the body, and therefore excreted unchanged, has received no support from any recent chemical or physiological discoveries. Nor can we say any more in favour of the doctrine propounded by Dr. P. W. Latham, that diabetes is due to imperfect oxidation of the muscle-albumin and the formation of sugar by the condensation of six molecules of methyl aldehyde. Dr. Pavy's latest researches have left his improved hypothesis of vasomotor paralysis of the vessels in the splanchnic area exactly where it was; its place in the nervous hypothesis is now occupied by the conception of an excito-secretory nerve which controls the sugar-forming function of the liver, and which, as we have already seen, may be affected by various stimulating or depressing agencies.

Morbid Anatomy.—The bodies of persons dying from diabetes present

as many departures from the healthy standard as might be expected in the case of a disease in which nutrition is so gravely impaired; but most of these changes are to be regarded as simple failure in the reparative processes of the tissues.

External Appearances.—There is generally wasting and sometimes extreme emaciation; but in other cases a well-developed layer of subcutaneous fat may be present. The skin is thin and harsh, the hair scanty and dry, the teeth very defective, and there are frequently traces of rashes, or scars of boils and carbuncles.

Brain.—This organ presents no constant lesion, but was normal in eight only out of thirty-one cases under my own observation. The most common change is congestion and oedema, with thickening of the membranes. It is less often anaemic; sometimes the convolutions are wasted and the sulci widened. The brain tissue may contain cavities which have been aptly compared to those met with in Gruyère cheese; they vary in size from a pin's head to a horse-bean, and seem to depend upon local atrophy from failure of nutrition. The lateral ventricles and the iter have been found dilated without any obvious mechanical cause. The choroid plexuses may be congested or thickened, or may contain cysts. Of much greater importance are the tumours of the fourth ventricle and medulla, of which ten cases have been collected: in many of these there can be no reasonable doubt that the diabetes depended directly upon the growth. In the same situation also have been observed examples of softening, of sclerosis, of alterations of colour and congestion, and of the presence of corpora amylacea and colloid masses. The softening or sclerosis in this position may undoubtedly cause diabetes, but the other lesions are of more doubtful value. The enlargement of the perivascular spaces, to which Dickinson drew attention, is only a consequence of the failure of brain nutrition, as in the case of the cysts already described. Extensive haemorrhage into the brain is rare. Glycogen is present in large quantities in the medulla oblongata and in the sheaths of the vessels of the cortex. In the microscopical examination of numerous specimens by myself no special or characteristic structural changes could be detected in any part of the brain. No minute haemorrhages were found, and the cysts already mentioned shewed no trace of haematoidin-staining. In only one instance did the capillaries of the vagal nuclei seem to be abnormally numerous and full of blood.

Spinal Cord.—A certain number of cases of diabetes undoubtedly occur from the extension of disease from the cord into the medulla, and when this happens changes characteristic of locomotor ataxia, insular sclerosis, or like lesions, are present. We do not possess abundant materials for making very positive statements as to the general condition of the cord, for examination of it has often been omitted. The recorded facts point to the frequency of secondary nutritive changes; for example, dilatation of the central canal, enlargement of the perivascular sheaths, and localised softening. Some recent observations by Sandmeyer and by Dr. Williamson

have indicated that slight atrophic changes may occur in the posterior columns, chiefly marked by their failure to react normally to staining agents, although no symptoms of tabes dorsalis may have been present during life. A case of tumour of the cord followed by diabetes was one of myxoma of the dura mater recorded by Dr. Shingleton Smith. Glycogen is present in large quantities in the spinal membranes and sheaths of the vessels.

Cerebrospinal Nerves.—Three examples have been collected of diabetes caused by tumours growing from the right vagus ; though in one instance (that of Frerichs) the growth encroached upon the floor of the fourth ventricle. Lubimoff has also described a case of diabetes in which atrophy and pigmentation of the inferior ganglion of the vagus were present. The peripheral nerves may shew interstitial inflammation, characterised by great increase of connective tissue, with secondary destruction of the axis-cylinders ; and this change may cause well-marked symptoms. The nerves of the lower extremities are chiefly affected, but any part may be attacked.

Sympathetic Nerves and Ganglia.—Changes in these structures early attracted the attention of students of the pathology of diabetes, as they occur with some frequency. Duncan, in 1818, found the sympathetic trunk in the abdomen three times as thick as normal. Percy, in 1842, described the semilunar ganglia, the splanchnic nerves and vagi, as thickened and of cartilaginous hardness. Lubimoff found sclerosis of the sympathetic ganglia and atrophy of their nerve-cells. Cavazzani has described atrophy of the coeliac plexus. The semilunar ganglia in my cases have been four times enlarged, once atrophied, and once embedded in a mass of fibrous tissue ; but, as a rule, they were normal, and the histological changes the same as are met with in many other maladies, although these ganglia are never so greatly enlarged as in diabetes. In spite, therefore, of the extent of these changes, they are not to be regarded as the cause of the disease.

Heart.—The myocardium is often pale and soft, more rarely hypertrophied or distinctly fatty. Pericarditis occurs occasionally and endocarditis sometimes, while coincident valvular disease may be present. Advanced fatty change of the muscular fibres is the characteristic change in old-standing cases of diabetes. Glycogen granules may be found between the muscular bundles.

Blood.—The appearance of the blood is generally normal ; but at times it is loaded with fat, which, after the blood is shed, floats on its surface in a cream-like layer. Under the microscope this fat is seen to be present as a very fine emulsion, but the granules run together after death to form droplets, and thus give rise to the appearance of fat-embolism in the capillaries : this, however, is but a post-mortem change. The red corpuscles are sometimes broken down into a granular material, and they are generally reduced in number. There may be an abnormal abundance of myelocytes or of large mononuclear leucocytes.

Chemically the blood contains, as a rule, more sugar than it does in

health, and the proportion may be as high as 4 per mille. The alkalinity of the blood-serum is reduced in consequence of the presence of acids of doubtful identity, β-oxybutyric acid and diacetic acid being the most probable. The presence of acetone, which may be due to the splitting up of diacetic acid into acetone and CO_2, has been observed, but is not constant even after death from coma.

Lungs.—Secondary changes in these organs are very common, the most usual being congestion and oedema. Next in frequency comes phthisis, usually but, as proved by the case recorded by Roque Dévic, not invariably tuberculous. Lobar pneumonia occurs rarely, but is very acute and fatal; acute bronchopneumonia is sometimes met with. Small foci of softening, abscesses, haemorrhagic infarcts, and gangrene may be found. Pleurisy and empyema occur sometimes. Fat-embolisms of the pulmonary capillaries have been described, but these are really post-mortem changes in the fatty blood. Hyaline fibroid thickening of the vessels occurs as part of the tissue-changes of chronic inflammation.

Liver.—This organ is generally enlarged, weighing from 60 to 80 ounces; less commonly it is small, pale, and soft. It is sometimes fatty, often congested, and its consistence may be abnormally firm. It frequently presents a certain amount of interstitial hepatitis, and occasionally it may be distinctly cirrhotic. The new growth commences in both the hepatic and portal areas. This form of cirrhosis is sometimes associated with haemochromatosis and bronzing of the skin (Hanot and Schachmann). Abscess of the liver is occasionally met with in cases of diabetes, and then is probably a causal lesion. Dickinson has described thrombosis of branches of the portal vein, and angiomas formed of dilated capillaries near the radicles of the hepatic vein. According to Weyl and Apt the diabetic liver does not contain excess of fat, and absence of this substance from the hepatic cells has been noticed by Beale and Frerichs. Quincke thinks that iron is in excess, but Zaleski estimates it at 0·685 per mille, and points out that we have no data to enable us to say whether this amount is greater than normal or not.

Spleen.—This organ is commonly said to be small, pale, and soft: less often it is enlarged and congested; sometimes it contains tubercle; glycogen has been found here as elsewhere. Hyaline degeneration of the vessels has been described.

Pancreas.—Great interest attaches to the alterations in this organ, since the important researches of Minkowski have shewn that its complete destruction in dogs is followed constantly by all the symptoms of diabetes; and many papers have been published on the subject (Baumel, Churton, Vaughan Harley, Williamson, G. Hoppe-Seyler, Hansemann, Fleiner, and others). Unfortunately we have still a good deal to learn about the significance of slighter changes in its structure, and it is possible that, while appearing normal to the naked eye, it may be extensively diseased. Lancereaux thought that pancreatic disease was invariably associated with the clinical type which he called diabète maigre; and this association is undoubtedly the rule although, since attention has been drawn to the

matter, many exceptions have been recorded, and fat diabetics have been shewn to present well-marked pancreatic atrophy (Baumel, Lépine, G. Hoppe-Seyler, Williamson). As to the relative frequency of these changes my notes of 27 consecutive cases give the following results :—

Pancreas atrophied	13
,, large and hard	5
,, large and soft	1
,, large	1
,, congested (mottled)	1
,, normal	6
	27

In two cases reported by Fleiner attacks of pancreatic colic had preceded for years the onset of diabetes, which, in fact, did not supervene until cirrhosis had destroyed the entire gland. Several cases of cancer of the pancreas associated with all the symptoms of diabetes have been published. Other cases are on record of diabetes with cystic disease of the pancreas and with multiple pancreatic abscesses.

In the morbid specimens examined the lesions described in my Bradshaw lecture (1890) have always been found, namely, varying degrees of interstitial inflammation with formation of large areas of connective tissue and new ducts. Stress has of late years been laid on the destruction of the so-called islets of Langerhans as essential, especially by Opie, who has described hyaline degeneration of the islets as the sole lesion in a case of diabetes ; but Dr. Dale has proved that these "islets" are merely phases in the life-history of the secreting alveoli, and are formed from the latter as the result of activity : in other words, they are not independent structures. When the pancreas is stimulated to exhaustion by secretion there is a great formation of islet tissue from the secreting alveoli ; nothing is known at present as to the relations, if any, which these islets bear to carbohydrate metabolism. In the earlier stages of the process the gland becomes swollen and infiltrated with small round cells. Besides this cirrhotic atrophy the gland may undergo fatty degeneration, which leads to complete transformation of its entire substance into fat. It has been suggested that the changes in the pancreas are secondary to disease of the coeliac plexus, but Lustig and Peiper have shewn that extirpation of this plexus is not followed by atrophy of the pancreas.

Stomach.—Although our data are by no means complete on this part of our subject, this viscus is seldom found to be normal. It is often dilated, and shews evidence of chronic gastritis ; the mucosa may be congested or contain haemorrhages, and is not uncommonly thickened.

Intestines.—These share in the congestion and catarrh which are found in the stomach ; haemorrhages may be present in the duodenum. The large intestine is generally filled with hardened faeces, and occasionally shews dysenteric inflammation or desquamation of its epithelium.

Kidneys.—Although changes in these organs are undoubtedly secondary and variable in kind and degree, they are always present. They generally consist in enlargement and slight fatty change; less commonly congestion is observed. Sometimes the cortex is thinned and the organ contracted. Tuberculosis, lardaceous disease, and even gangrene have been found. But the only distinctive lesion is the hyaline transformation of the epithelium of Henle's tubes first described by Armanni, and named after him. It is not constantly present, and its etiology is still obscure. Glycogen may be found in the renal epithelium, as elsewhere, and marked fatty change may be present. Not uncommonly the kidneys exhibit all the characteristic histological appearances of chronic diffuse nephritis.

Bladder.—This viscus is usually normal, but may be dilated and hypertrophied, while its mucous lining may be the seat of haemorrhages or catarrhal changes.

Summary.—Diabetes is a disease which has so profound an influence upon the general nutrition of the body, that it tends to produce structural alterations in the various organs, which are for the most part of a secondary and degenerative character. The exceptions are (i.) the tumours and growths in or near the medulla oblongata and the vagi nerves; (ii.) a few instances of primary liver disease; (iii.) cirrhosis and other destructive changes in the pancreas. The one important addition to the morbid anatomy of diabetes which the last few years have yielded is undoubtedly the lesions of the pancreas; and we are justified in regarding these changes, when present, as the cause of the symptoms in the same sense as granular kidney is the cause of the symptoms of chronic Bright's disease.

Symptoms and Course.—Diabetes manifests itself in two principal clinical forms—(*a*) acute and (*b*) chronic, which differ in the intensity of the essential symptoms: these are glycosuria, polyuria, thirst, and wasting—symptoms which are always excessive in the acute and more moderate in the chronic form. Acute diabetes usually occurs in persons under forty years of age, and not uncommonly in children or young adults. The patient complains of weakness, of thirst, and of passing an excessive quantity of water. The frequency of micturition interferes greatly with sleep, and the want of rest is one of the chief causes of the great constitutional depression. In spite of an appetite which may be voracious the body-weight diminishes rapidly; the face is often flushed, the skin dry, the hair rough, the lips parched, the tongue red and sticky, or covered with a black fur, the secretions of the mouth diminished, and the bowels confined. There is often a persistent nauseous sweet taste in the mouth, the breath may have a sweet odour; the muscular strength is much impaired; sexual appetite is usually lost, and the mind is depressed. The quantity of urine varies from five to fifteen pints or more; it contains from 5 to 10 or 12 per cent of sugar, and gives the ferric chloride reaction.

Chronic diabetes, on the other hand, occurs as a rule in elderly people of both sexes, and often in those who are or have been decidedly obese.

Such patients complain of weakness, of frequent micturition leading to disturbed nights and of some loss of flesh; sexual desire is generally absent; the mind is often depressed, and the digestive organs are disturbed. The quantity of urine may vary from three to six pints, the sugar from 3 to 10 per cent, and the ferric chloride reaction is generally absent.

There is nothing constant in the external appearance of a diabetic patient; although the peculiar flush which, when present, is not limited to the malar eminences but resembles a deep blush extending up to the roots of the hair, may suggest to an experienced observer the true nature of the case. The odour of the breath is also significant. The nutrition of the skin and its appendages suffers constantly; so that the epidermis becomes dry and rough, the nails brittle, and the hair thin and dry. The temperature of the body is usually subnormal, but an increase has been observed at the onset of some acute cases reaching as high as 103° F. (39·4° C.).

As already mentioned there may be mental depression or irritability of temper; vision is frequently impaired, the other senses less commonly so; common sensibility to touch and pain, and sensations of heat and cold, remain normal; neuralgic affections are often observed, and occasionally there may be paralysis or ataxy from peripheral neuritis. The knee-jerks are sometimes diminished or lost, but are generally normal. In women menstruation is as a rule deficient or absent. The appetite is usually good and digestion easy; but the bowels are almost always confined, though in some cases there is a marked tendency to diarrhoea. The teeth are very often decayed or falling out from atrophy of the gums. The stools have a peculiarly fetid odour. The cardiac impulse is usually normal in position, but in advanced cases it is diffused and weak. The pulse, which at first shews a tracing of high pressure, becomes, in the later stages, small and feeble, but its rate is not increased except in consequence of some complication. The examination of the blood-pressure of 15 cases of diabetes with Riva-Rocci's instrument has shewn that, except in cases complicated by albuminuria, the blood-pressure is never higher than 140 mm. of mercury. On the other hand, in severe cases it may be as low as 110 or 115. A rapid pulse is one of the early signs of the onset of coma.

The blood contains excess of sugar, and often a marked increase of fat; its alkalinity may be reduced 50 per cent, but its microscopic appearances are generally normal. In advanced cases there may be a great reduction of the red blood-corpuscles (50 per cent), and it has been asserted that they do not stain normally with eosin (Bremer), but this change is certainly not constant. The serum is poisonous to rabbits (Roque Dévic). Sugar may be present in the sweat, tears, and saliva. The body-weight often undergoes rapid reduction before the institution of proper treatment, but after this has commenced it may vary from day to day within three or four pounds, although there may be no constant progress in gain or loss.

The persistent presence of sugar in the urine is the cardinal sign of the disease, and we will therefore inquire whether glycosuria ever occurs apart from diabetes. Traces of sugar, as we have seen, may be found in normal urine; but in the urine of non-diabetic patients it is rare to meet with a quantity sufficient for demonstration by the ordinary tests. It has been stated that the urine of healthy breast-fed infants contains a substance which reduces cupric salts, but does not ferment. So-called "alimentary glycosuria" sometimes occurs in persons who have taken much sugar in food or drink, and it remains an open question whether such an occurrence indicates a tendency to diabetes. I have recorded an instance of persistent glycosuria in a man who appeared quite healthy, and ten years after I first found sugar in his urine, he was in good health, although Fehling's solution was still reduced by his urine to a degree indicative of 1·2 per cent of sugar. This condition, I have now discovered, was due to alcoholism. Sugar is frequently, if not constantly, found in the urine of persons who have been drinking heavily, apart altogether from the presence of sugar in the drink, but it passes away rapidly. This is a common cause of mistakes in diagnosis, as the sugar persists so long as the alcoholic habit is maintained.

Temporary glycosuria occurs in many surgical conditions, after injuries, in some nervous diseases, in gout and other general maladies. However, under the heading of the various tests for sugar, certain fallacies will be pointed out which in the past have led to many erroneous statements of the presence of sugar in the urine.

Urine.—The urine is generally greatly increased in quantity, but varies within wide limits; when diarrhoea is persistent it may be normal or even less than normal in amount. The specific gravity of the urine is usually high, varying from 1025 to 1050; but a low density does not exclude sugar, nor a high density prove its presence. In some of my cases it has been as low as 1013, while phosphatic urine may be as high as 1040. The colour in typical specimens is pale greenish-yellow, but it varies through all shades of yellow up to deep amber; the liquid is generally clear, but may be turbid from lithates, muco-pus, or torulae. It does not tend to decompose so readily as normal urine, but affords a favourable medium for the growth of the yeast plant. Its reaction is almost invariably strongly acid; and it deposits very commonly a considerable amount of uric acid crystals. In women vaginitis is often present, so that the urine may contain pus and epithelium. The normal constituents are increased; there is an absolute excess of water, chlorides, sulphates, creatinine, ammonia, phosphates, and urea. The phosphates and urea usually bear the proportion to each other of one to ten (Butel); and the urea to sugar of one to two (Harrison and Slater), but there are many exceptions to this rule.

When carbohydrates are added to the diet the amount of sugar increases, but the urea diminishes. The loss of lime in combination as phosphate is very great, and the amount even approximates to that observed in mollities ossium. Some recognise a phosphatic diabetes

(*vide* p. 228), and cases occur in which phosphaturia precedes or alternates with glycosuria. The amount of ammonia eliminated is very large (Hallervorden), and this is attributed by Stadelmann to the excess of acid in the blood, which disturbs the normal mechanism for fixing ammonia; he therefore regards it as an index to the blood condition and a premonitory sign of diabetic coma.

The *amount of sugar* may vary up to 12 per cent. In some rare cases *air* has been found mixed with urine on its passage from the bladder, and it has been suggested that this is due to fermentative decomposition of sugar in the bladder with formation of CO_2, but the presence of alcohol has not been demonstrated. *Glycogen* is said to be constantly present, and *laevulose* has been met with. In some cases the sugar may disappear for a time and then return. *Albumin*, if present, may be due to an admixture of discharges, as of vaginitis and vesical catarrh, or to the presence of chronic nephritis. It is less common in young subjects; it is generally but not constantly present in the urine of cases of death from coma. Indican and skatoxylsulphuric acid may be present in excess; acetone, aceto-acetic acid, and β-oxybutyric acid, and crotonic acid—all chemically related substances—have been found, and have excited much interest, as each or all of them has been supposed to be the poison which causes Kussmaul's coma; and some of them give the well-known Burgundy-red coloration with ferric chloride. This reaction is also given by formic acid, which is alleged to be sometimes present. Oxalic and hippuric acids frequently occur.

Tests for Sugar.—Fermentation was the earliest method devised to prove the presence of sugar in the urine, and it is still the best; but it is too tedious to be useful on all occasions, and some little care is needed to demonstrate the presence of small quantities. Under the influence of the *Torula cerevisiae*, or beer-yeast, sugar splits up into ethyl alcohol and carbonic anhydride, the process taking place best at 35° C. (95° F.) The amount of sugar may be estimated by the diminished specific gravity of the urine, or by collecting the CO_2; one molecule of grape-sugar giving two molecules of CO_2. For this a volumetric analysis or special fermentation-tube is necessary; but for qualitative testing an ordinary test-tube filled for two-thirds of its depth with mercury, and inverted over the same metal, will answer the purpose. A little tartaric acid should be added to the urine, and the yeast should be well washed. Fermentation is ordinarily effected by taking two separate specimens of the urine, adding yeast to one, and putting both for some hours in a warm place; after fermentation has taken place the densities of the two specimens are compared, and the amount of sugar is calculated according to Roberts's formula, by which for each degree of density lost is reckoned one grain of sugar per ounce. The method is very fairly exact.

The usual qualitative test is Fehling's solution. This is prepared by mixing equal quantities of two liquids, (*a*) and (*b*). (*a*) consists of pure sulphate of copper (34·64 grammes) dissolved in distilled water (to 500 c.c.); (*b*) consists of pure sodium potassium tartrate (173 grammes) dissolved

in a solution of caustic soda (100 c.c.) of sp. gr. 1·34, and diluted with distilled water (to 500 c.c.) On mixing exactly equal quantities a clear deep blue liquid is found, of which 10 c.c. = 0·05 gramme of dry diabetic sugar. As the mixture is liable to decompose, the liquids are better kept separate until required for use.

To determine the presence of sugar, about a drachm of the mixture, and an equal quantity or less of the urine, should be thoroughly boiled together. If sugar be present the cupric is reduced to cuprous oxide, and the yellow suboxide of copper is thrown down.

This test is unfortunately not absolutely certain, as the urine may contain other reducing substances, of which glycuronic acid, uric acid, creatinine, and lactose are well known. The urine of persons taking salicylic acid or its salts, chloral, chloroform, lactic acid, or aldehyde may give this reaction. It is therefore often of great importance to employ a confirmatory test, especially where only a trace of sugar appears to be present. Many exist, but the best is that devised by von Jaksch, which depends upon the property of phenyl-hydrazine to form with grape-sugar a characteristic crystalline compound called phenyl-glucosazone. Two parts of hydrochlorate of phenyl-hydrazine ("twice as much of the salt as will lie on the point of the blade of a knife") and three of acetate of soda are placed in a test-tube containing three or four drachms of urine; if the salts do not dissolve when the fluid is warmed a little water is added, and the tube placed for twenty to thirty minutes in boiling water. It is afterwards taken out and allowed to stand in cold water, when, if sugar be present, a yellow precipitate is formed at once which, under the microscope, consists of detached or radiating clusters of characteristic yellow needles. If albumin be present it is better to get rid of it by previous boiling and filtration.

Quantitative estimation is usually performed by Fehling's solution, of which 10 c.c. = 0·05 gramme of grape-sugar. To perform the estimation we require a burette graduated in cubic centimetres, a hundred-cubic-centimetre glass measure, a porcelain capsule, an iron tripod, and a spirit lamp or Bunsen burner. Ten c.c. of Fehling's solution are measured off, diluted to 50 c.c. with distilled water, and placed to boil in the capsule. Ten c.c. of urine are now measured off, diluted and well mixed with nine volumes of distilled water; the burette is then filled with the diluted urine to zero on the scale. When the Fehling's solution begins to boil the diluted urine should be run into it drop by drop, the operator constantly stirring the mixture, and carefully watching the result until the blue colour entirely disappears, and the whole of the copper is reduced. The calculation is then readily made thus:—Suppose that 20 c.c. of diluted urine have been used to reduce 10 c.c. of Fehling, which is equal to 0·05 grm. grape-sugar, the percentage is $\frac{0{\cdot}05 \times 100}{20} = \frac{5}{20} = 0{\cdot}25$; but as the urine was diluted nine times, $0{\cdot}25 \times 10 = 2{\cdot}5$ per cent. The method does not pretend to perfect accuracy, but it is quite sufficient for clinical purposes.

Grape-sugar deflects polarised light to the right hand, and upon this

is based a method of estimation by means of a somewhat expensive instrument called a polarimeter. The operation needs some care and practice; but a number of experiments, made by Dr. S. H. Perry in my wards, shewed that it may afford very uniform results, although these were constantly less than the figures obtained by fermentation and Fehling's method. This variation may have been due to the presence of other substances, such as laevulose and β-oxybutyric acid, which rotate light in the opposite direction, and consequently diminish the net amount.

Among other tests for sugar is Moore's, in which the urine is treated with liquor potassae, and boiled; if sugar be present it is decomposed, and imparts an intense brown coloration to the fluid; but mucin and bile give the same reaction, and the alkali used must be free from lead, hence this test alone is not trustworthy nor is it very delicate. Nylander's test is performed by mixing a quantity of urine with an equal volume of the following solution:—Bismuth subnitrate, 2 grms.; potassic sodic tartrate, 4 grms.; caustic soda (sp. gr. 1·12), 100 grms. The mixture is then shaken and boiled. In the presence of sugar a black deposit is formed from the reduction of bismuth oxide. It is less sensitive than Fehling, and this precipitate occurs in the urine of persons taking rhubarb, and in the presence of albumin, pus, blood, or mucus. The test devised by Braun, and recommended by Johnson, is performed by adding to a drachm of urine ten drops of concentrated solution of picric acid, half a drachm of liq. potassae, and enough water to make up two drachms. On boiling the mixture it becomes a deep reddish brown if sugar be present; but in the absence of sugar a certain amount of the same colour is often produced, so that Johnson holds that the fluid must become opaque; herein lies a doubt in many cases.

The indigo-carmine test first proposed by Mulder is neither sensitive nor accurate; the urine is treated with solution of sodium carbonate, and solution of indigo-carmine is added until the whole is freely coloured; on heating the colour changes to yellow in the presence of sugar, and becomes blue again on being shaken up with air.

Krismer's test is performed by taking equal parts of urine, liquor potassae (B.P.), and solution of safranine (1 to 1000), and heating the mixture, which decolorises in the presence of sugar. The reaction is very delicate, but occurs very frequently when no sugar is present. It is said that some specimens of safranine act better than others.

Rubner's test for sugar is made by adding to 10 c.c. of urine an equal quantity of neutral acetate of lead, and filtering. To the filtrate ammonia is added drop by drop until a thick, curdy precipitate falls; this must be heated cautiously up to 80° C. (176° F.), when if grape-sugar be present it turns rosy red; on further heating it becomes coffee-brown. The reaction will indicate the presence of 0·25 per cent of sugar.

Acetone may be most readily detected in the urine by Le Nobel's method. A few drops of a weak solution of nitro-prusside of sodium (1 per cent) are added to the urine, followed by excess of ammonia, the

mixture turns to an amethyst colour in the presence of acetone; but it requires some minutes to change. On boiling and acidulation this colour changes to greenish blue.

In Legal's test caustic potash takes the place of ammonia, and the resulting colour is red, which disappears on standing; on adding acetic acid a deep violet colour is produced.

Lieben's test, as modified by Ralfe, is performed by floating a drachm of urine upon a drachm of liq. potassae containing 20 grains of iodide of potassium, and previously boiled in the test-tube. In the presence of acetone the phosphatic ring, which forms at the point of contact, turns yellow, or is studded with yellow points from the formation of crystals of iodoform; this reaction, however, has the disadvantage that it is produced by lactic acid and alcohol as well as by acetone.

The ferric chloride reaction is the name given to the Burgundy-red coloration assumed by urines on the addition of a few drops of liq. ferri perchloridi. It is probable that the cause of this reaction in diabetic urine is the presence of aceto-acetic acid, or diacetic acid as it is commonly called, an acid which readily breaks up to form acetone and carbonic anhydride; but many other more or less nearly allied substances also give this reaction, and the urine of patients taking salicylates or salicylic acid, antipyrin, thallin, and allied compounds which give a deep purple, may cause confusion. The characteristic colour is a deep rich Burgundy or claret colour. The reaction of diacetic acid may be distinguished by boiling another specimen of the urine for some time before applying the test; as this acid is driven off by heat the reaction will be modified. This reaction is not peculiar to diabetes, but is of considerable practical importance as roughly serving to distinguish the severe cases from the mild. It may be safely accepted that cases which shew this reaction are not curable, and their treatment should be entered upon with caution.

Von Noorden attaches great importance to the presence of β-oxybutyric acid in the urine, for he regards this substance as the sole cause of diabetic coma; although Roque Dévic found none in the very careful examination he made of his case. The determination of the presence of this acid can only be performed with certainty by a difficult and prolonged chemical process (Wolpe, Külz); but von Noorden suggests that its presence may be inferred with reasonable probability when the urine is laevo-rotatory after titration with Fehling, fermentation, or precipitation with basic acetate of lead and ammonia.

Duration.—As a general rule diabetes in children and young persons is an acute and rapidly fatal disease, lasting only weeks or months, or at most one or two years; but even in children cases have been known to extend over five, six, or more years. On the other hand, in elderly people the disease usually makes but slow progress, and lasts many years.

Termination.—Death occurs in many cases as a result of one or other of the complications to be described, of which pulmonary phthisis is perhaps the most common. Very often the patient's strength becomes

gradually diminished, and he dies quietly in a drowsy condition without actually becoming comatose. But in many cases the accidents that precede death come on more or less suddenly, from some slight cause, such as fatigue, excitement, or a chill; and death is preceded by coma of a peculiar type. Any acute infectious process is peculiarly liable to terminate fatally in diabetes; this has been abundantly illustrated in the recent years owing to the great prevalence of influenza, which has proved fatal to many diabetics, even where no special visceral complications, such as pneumonia, have been manifested; as a rule these cases have died comatose.

Complications.—*The Skin.*—Diabetics suffer from many derangements of the skin besides the dryness and roughness of the epidermis which are almost always present. The circulation is feeble; the ears, nose, and cheeks are often cyanosed, and the legs and feet are cold. Elderly patients are peculiarly liable to acne pustules, boils, and carbuncles; and a papular erythema on the extremities is one of the common ailments of younger subjects. This last presents rose-coloured, elevated spots, as large as a split-pea, thickly distributed over the elbows, knees, wrists, ankles, fingers, and palms of the hands; these often coalesce to form red shiny patches.

Symmetrical erythema may attack the face. Davies Pryce has described an erythematous oedema, which depends, as he believes, on the presence of neuritis. The palms of the hands and the soles of the feet may burn intensely or sweat profusely. The occasional occurrence of general sweating has been already noticed.

Eczema of the genitals, especially in women, may be a most distressing symptom. The disease is undoubtedly set up by the irritation produced by torulae and other organisms which grow in the saccharine moisture remaining on the parts; but it often results in an intense dermatitis which spreads over the abdomen and thighs, and itches intolerably. It may be prevented by scrupulous cleanliness. Pruritus vulvae is sometimes present without dermatitis. Purpura may be seen in the early stages.

A horrible affection, described by Kaposi, and called by him papillomatosis diabetica, occurred in the person of a Brazilian patient, and was probably caused by some exotic parasitic growth; the hands and forearms were covered with ulcerating excrescences and warty growths.

Xanthoma diabeticorum, originally described by Addison and Gull, consists of indurated, rounded, or conical tubercles of a dull reddish colour, their apices being often yellow. The yellow colour is due to the presence of fat-globules. Marchal (de Calvi) relates a case in which the body of the patient was covered with great coppery pustules containing materials as hard as very dry cheese. It disappeared, leaving only minute scars, and did not recur. This appears to have been a case of xanthoma, which tends to disappear in the same way.

Little centres of necrosis in the skin about the ankles and dorsum of the feet, first appearing as small round red spots the size of pin-heads, have been described. The disease seems to begin in the sweat-glands.

Cellulitis and gangrene are more apt to occur in diabetes as life advances, and are more frequent in men than in women. Gangrene sometimes depends on vascular disease, necessitating amputation high up; in other cases it is due to neuritis, and may be let alone, or the necrosed parts only removed by amputation.

The condition called by Kaposi gangrena bullosa serpiginosa was probably due to neuritis. The patient, a woman aged 61, had three gangrenous patches on her left leg, and fifteen or twenty bullae distributed over the neighbouring skin. Perforating ulcer, generally admitted to be due to neuritis, is not uncommon; it is preceded by a circumscribed anaesthetic patch, and when formed the ulcer is surrounded by an anaesthetic zone.

Oedema of the subcutaneous cellular tissue of the lower extremities is sometimes present, and ascites may occur. In the cases of this kind which have come under my observation the dropsy has been due to heart failure, possibly with latent valvular disease.

The temperature in the axilla may be very low. Fagge recorded it as low as 93·6° F.; and in one of my own cases it was only 95° in the morning, though in the evening it used to rise to 100°.

Nervous System.—It is not uncommon to meet with listlessness and depression of spirits, weakness of mind, and peevishness of temper; definite mental disturbances, as indicated by melancholia with suicidal tendencies or temporary mania, occur more rarely. In some cases the mental disturbance and glycosuria have appeared to alternate. There may be symptoms resembling those due to an intracranial growth; for example, headache and giddiness, epileptic or apoplectic attacks, paralysis of the sixth pair with conjugate deviation of the eyes, ptosis, strabismus, or paralysis of a limb, or complete hemiplegia.

Neuralgia is usually symmetrical; it may come on suddenly, after the patient is in bed, the pain being excruciating. Each attack lasts a few hours only, but it may recur at short intervals. Sciatica is very common, and bilateral sciatica is especially suggestive of diabetes.

Some patients present symptoms resembling tabes, with ataxic gait, loss of knee-jerks, gastric crises, and lightning pains in the legs. These symptoms are often associated with sciatica, and depend upon a neuritis affecting the sciatic nerves and their branches. The pupillary reflex was thought to be never abolished, but Grube has shewn that this is unfortunately not invariably true, so that the differentiation from true tabes becomes very difficult. The soles of the feet may be intensely hyperaesthetic, and the other phenomena of neuritis may occur, such as hyperidrosis, glossy skin, oedema, ecchymoses, perforating ulcer, loss of nails, and so forth.

Eye Affections.—Diabetes causes impaired vision most commonly by weakening the power of the muscles of accommodation; and in the next place by diminishing the perception of light in the retina—diabetic amblyopia. After these changes, in order of frequency, comes cataract, which is usually but not always of the soft variety, and may disappear

spontaneously. The retina is liable to be the seat of a peculiar inflammation which takes two principal forms; the cases may be classified as—(i.) Retinitis centralis punctata diabetica; (ii.) Retinitis haemorrhagica diabetica; (iii.) Mixed forms. In the first the ophthalmoscope shews white glistening patches, not necessarily arranged round the macula, and never fan-shaped, but otherwise bearing a considerable general resemblance to albuminuric retinitis, with which, however, it must not be confounded: in the second the lesion consists of rounded or punctiform haemorrhages with secondary retinitis; and in the third we have a combination of both changes. Central scotoma, or loss of vision in the central part of the field, is common; and exactly resembles the condition met with in tobacco-smokers. It is probable that tobacco may produce its toxic effect upon the retina more readily in diabetes, but undoubted cases observed in non-smokers have been recorded. The optic nerve is sometimes atrophied, and capillary aneurysms on the retinal vessels have been described. Diabetics also suffer, as I have said, from paralysis of the ocular muscles; and hemiopia, various inflammatory affections of the ocular structures, conjunctivitis, keratitis, iritis, and choroiditis are met with.

Other Special Sense Affections.—Blunting of the senses of smell and taste has been recorded in one case. Deafness, due to otitis media, or to oedematous swelling of the Eustachian lining, may occur. Diabetic otitis media is a very acute inflammation, coming on suddenly without any previous coryza or cold; it is characterised by severe external pain in the mastoid region, tinnitus, and extreme deafness. The auditory canal becomes red and swollen, secreting muco-pus; the tympanic membrane is congested, oedematous, and dull; the mastoid cells are infiltrated with pus, and the osseous tissue may be extensively destroyed. According to Raynaud, the disease begins in the bone. Fever may be entirely absent. In some cases true nerve deafness has been observed.

Respiratory System.—A case of acute and rapidly fatal membranous inflammation of the larynx and trachea attacking a male patient under treatment for diabetes in the Royal Infirmary of Edinburgh was reported by Warburton Begbie; but with this exception no mention of any complications affecting these structures has come under my notice, and it is possible that this was merely an example of diphtheria occurring in the course of diabetes. The bronchial tubes may be affected by catarrh, and this may precede the appearance of pulmonary phthisis, which is one of the most common complications. Since the discovery of the tubercle bacillus has placed within our reach an easy means of determining whether a pulmonary lesion be tuberculous or not, it has become clear that non-tuberculous cases are much more rare than was formerly supposed; there can be no doubt, however, that they do sometimes occur. I have had three cases in which I could find no tubercle bacilli, but a more conclusive case with careful post-mortem examination has been recorded by Roque Dévic and Huguenenq. The disease comes on as a rule insidiously, and is often attended by little cough or rise of temperature. It may, however, follow an attack of acute lobar pneumonia. Such attacks of

pneumonia in diabetic patients often prove fatal, but by no means invariably so; complete recovery may take place, or permanent lung mischief may follow. Gangrene may supervene upon acute or chronic inflammation of the lungs, and is more often met with in diabetes than in any other constitutional state. It is often undetected during life, as the characteristic offensive odour of the sputa may be entirely absent.

Circulatory System.—Diabetic endocarditis is a rare complication; it most commonly affects the mitral valves, rarely those of the aortic opening. Heart failure, on the other hand, due to fatty and fibroid changes, is a common occurrence, and may be regarded almost as part of the ordinary course of the disease. It may be associated with attacks of dyspnoea or faintness, or of angina pectoris; and such symptoms should be regarded as indications of the danger of sudden death. It is therefore of great importance to watch the state of the circulation, and to observe the character of the pulse. Elderly patients are not exempt from the ordinary senile changes in the vessels, and these when seated in the extremities may lead to gangrene.

Digestive System.—Loosening and loss of the teeth, from atrophy of the gums, gingivitis, and spongy or bleeding gums, are common; the teeth themselves are also very apt to be attacked by caries, causing toothache, abscess of the root, and the like. Loss of sleep from toothache is a very serious matter to a diabetic, and careful attention to the hygiene of the mouth is most important. The saliva is generally diminished, so that the mouth is dry, and sour eructations are not uncommon. Hunger is one of the symptoms of diabetes, and sometimes a feeling of emptiness at the pit of the stomach is troublesome. As a rule diabetic patients do not suffer from painful digestion, but the tongue is often furred, and post-mortem examination shews the frequency of gastritis. Some patients are liable to periodical attacks of gastritis, which may be attended by jaundice or diarrhoea; and according to Grube gastric crises, resembling those of tabes, are by no means rare. Examination of the stomach functions does not indicate that there is any greater disturbance of the digestive capacity than might be expected to result from the chronic gastritis. Hirschfeld has described cases which were characterised by early attacks of pains, with light-coloured, fatty stools, suggestive of pancreatic colic; and these may be compared with Fleiner's cases, already quoted, in which diabetes supervened years after the attacks of pancreatic colic. Diarrhoea is sometimes very troublesome, needing considerable care in diet. Unfortunately diabetic diet is not very digestible; the bran, almond or cocoa-nut biscuits, green vegetables and salads, prescribed for these patients as substitutes for bread and potatoes, contain a large amount of indigestible vegetable material which acts as a mechanical irritant to the gastro-intestinal mucosa. In spite of this constipation is the rule due to abnormal dryness of the intestinal contents.

Enteric Fever.—Diabetic patients appear to be more than ordinarily

susceptible to the poison of enteric fever, but the attacks are usually mild in type. During the fever the sugar generally disappears. Prof. Noel Paton has shewn that increased elimination of heat in animals favours glycogenesis, but that the products of the growth of micro-organisms inhibit it; to the latter, then, we must ascribe the disappearance of the sugar.

Rheumatism.—Rheumatic muscular pains are very common in diabetes, and thickening and shortening of the palmar aponeurosis (Dupuytren's contraction) has been repeatedly noticed; though it is probably due to a rheumatic or gouty condition of the patient and not to diabetes.

New Growths.—Some authorities believe that diabetes disposes to the formation of tumours, but that when they occur they grow slowly; the grounds for these opinions are not very clear.

Diabetic Coma.—The liability of diabetes to terminate suddenly in a peculiar form of coma has been known for many years, but its special features were first described in 1874 by Kussmaul, whose name has been attached to the affection. Attention to the phenomena in question was first aroused in England by Sir Walter Foster's paper at the Manchester meeting of the British Medical Association in 1877. Kussmaul's coma occurs in diabetics at all ages, and in both sexes, but with much greater frequency in the young. The direct causes are fatigue, excitement, exposure to cold, and any intercurrent acute disease. The remoter causes are absence of starchy food and constipation. Its pathology is still obscure; Kussmaul's original suggestion that it is due to poisoning by acetone has not been proved, and careful analysis has failed to detect this substance in the blood of some persons dying in this manner. The symptoms resemble those produced in animals by large doses of dilute acids, and considerable quantities of acid have been shewn to be present in the blood. This acid has been identified by Minkowski as β-oxybutyric acid, a substance which breaks up to form aceto-acetic acid, which, on further decomposition, yields acetone and carbonic anhydride. As I have already said, von Noorden regards β-oxybutyric acid as the true cause of the coma; but this can hardly be admitted in the face of the failure of Roque Dévic and Huguenenq to find any in the exhaustive examination of their case.

Dr. P. W. Latham suggested that the poison was paraldehyde, and even proposed a hypothesis of its formation; Senator has suggested trimethylamine, but unfortunately neither poison has been shewn to be present.

A few eminent authorities still regard the phenomena as uraemic; but, as the description will shew, the classical symptoms of uraemic coma are different in many respects. In diabetes the onset of coma may be preceded by malaise, languor, and weakness of some days' duration; there may be a gradual fall in the quantity and specific gravity of the urine, and this may give the reaction for acetone and with ferric chloride. Accelerated or disturbed respiration is a warning symptom which may be noted in advance by the physician. But in many instances the attack is

ushered in without previous warning by restlessness, delirium, or even maniacal excitement; or the patient may complain of weakness only. The pulse becomes very rapid and feeble, the epigastrium painful, and the respiration more hurried and deeper. In course of time drowsiness sets in, which deepens into coma; the patient then lies quietly in bed, breathing 30 or 40 per minute, with deep sighing respiration; the pulse beats 130 to 150 per minute, and is very feeble; the face is usually pale, the body and extremities cold, and the temperature subnormal. The excretion of urine is diminished or suppressed, and the bowels can be got to act with difficulty. Death is sometimes preceded by a rise in temperature (103°-104° F.), by convulsions, or by cyanosis. A peculiar odour of the breath is often present, but is not constant; it varies considerably in character, for it has been diversely likened to sour beer, rotten apples, hay, chloroform, and so on. The urine always contains acetone, and is generally but not constantly albuminous; the ferric chloride reaction is, as a rule, present, but exceptions to this rule have been observed.

The symptoms, as is indicated by the above description, though fairly uniform, do not always present the same picture; Frerichs endeavoured, not altogether successfully, to distinguish three groups, but it is doubtful whether there is either real utility or pathological justification for such a classification. It is of more interest to note that this form of coma is not peculiar to diabetes, but has been observed in pyonephrosis, chronic cystitis, gastric and hepatic cancer, anaemia, and other conditions.

Acetonuria is not of itself a certain indication of the imminence of this grave complication, as it has been often met with in patients who have lived for many months without such an occurrence. Hirschfeld suggests that in many cases this symptom is caused by the want of non-nitrogenous food, and that where this is the case it may be removed by the addition of a certain proportion of carbohydrates to the diet. He thinks this precaution should always be taken when acetonuria occurs, so that the occasional may be distinguished from the persistent cases, and also because the continuance of strict diet may in itself be a danger. His statements have been verified to a slight extent, but not sufficiently to enable us to assert that we have in this test a sure or even a useful means of prognosis. As will be seen when we come to speak of the treatment of diabetes, there is at the present time a salutary reaction against the too stringent diet of former years, so that there is now less probability of this kind of auto-intoxication arising from defective diet alone; and Kussmaul's coma has often come on in cases of diabetes in which, either by the care of the physician or by the wilfulness or neglect of the patient, a mixed diet had been taken. It is a very grave symptom, and although in view of the recoveries claimed by Reynolds, Quincke, Gamgee, and others it may not be invariably fatal, recovery is extremely rare. Less uncommonly we see the premonitory symptoms, such as epigastric pain, sighing breathing, and even some degree of drowsiness, pass away; in one case under my care this disappearance followed an attack of spontaneous diarrhoea,

and, besides this, another instance of recovery from epigastric pains and drowsiness is recorded in my lectures.

Prognosis.—Diabetes is in all cases a grave disease, and the subjects are regarded by all assurance companies as uninsurable lives: life seems to hang by a thread, a thread often cut by a very trifling accident. Still there is something to be said on the subject of prognosis besides the broad rules of age already laid down. In each case we must consider the general condition of the patient, the degree to which treatment is successful in reducing the sugar and maintaining nutrition, and the presence or absence of complications. The state of the circulation, as indicated by the pulse-pressure and cardiac impulse, is of great importance. Cases which supervene after injury or acute diseases sometimes get rapidly well, or recover more gradually, so that in these circumstances we may be hopeful at any rate for some time. Other points in favour of the patient are—(i.) the concurrence of obesity or gout; (ii.) favourable social conditions and freedom from business and financial worries; (iii.) early treatment.

Cases have ended in recovery in which the disease appeared to have been induced by eating excessive quantities of sugar. Such cases should probably be regarded only as temporary glycosuria; nevertheless the glycosuria often tends to recur in such persons, and in them there is probably some underlying tendency to diabetes. Schmidt considers that spontaneous sweating is favourable; but I have observed several instances which did not bear out his opinion. The cases which occur about the climacteric period in women end in recovery perhaps more frequently than other forms of diabetes in elderly people.

A good deal has been said of the prognostic value of the knee-jerks; some observers regard their absence as very grave, but extended experience has disproved this opinion, and the significance of the sign may easily be overrated (Grube).

On the other hand, where there is a marked family tendency to diabetes or to nervous disease, the ultimate prognosis must be unfavourable. The prospects of patients who are unable to diet themselves properly, to protect themselves from the vicissitudes of the weather, from the hardships of daily toil, or from the cares and anxieties of life, are undoubtedly less favourable; herein, as in most other chronic diseases, a competent income and a tranquil existence count for much in the prognosis.

Treatment.—Until the pathology of diabetes has been made perfectly clear, the best guides to treatment must be found in those generalisations from experience which have accumulated since the diagnosis has rested on a sure foundation. We have acquired much useful knowledge concerning the management of diet, the use of drugs, general hygiene, and so forth; but, above all, it is important to bear in mind that adherence to blind routine may be most disastrous, and that we are bound to study the needs of each individual case. Some patients are benefited by means which are harmful to others, and in many instances life may be shortened

by the vigorous application of rules which may be salutary for the majority. This may appear to be a self-evident proposition, but if so, there is none the less need to call attention to it, as the ordinary practice seems to be to copy a dietary out of a text-book, and hand it to the patient with a prescription of some preparation of opium, or of one of its alkaloids.

Where the store of glycogen in the liver is discharged under some temporary influence glycosuria results, but not diabetes; on the other hand where the causes persist, and all the sugar formed from the carbohydrates of the food continues to be poured directly into the circulation, the tissues become choked with sugar, and suffer in consequence a reduction of their sugar-consuming capacity. Under these last conditions the symptoms of diabetes appear, and can only be removed by suitable diet. The extent to which diabetes can be cured or alleviated depends upon the degree to which the sugar-consuming power of the tissues has been reduced. The object of treatment must be to relieve the tissues of the excess of sugar which is reaching them, and for this purpose we should give our patients a diet from which the sugar-forming elements are as far as possible withdrawn. In order to do this while yet affording the patient sufficient food to supply the needs of the body, the dietary should be constructed upon sound principles. According to Rubner, for each kilo ($2\frac{1}{5}$ lbs.) of body-weight there are consumed during repose in twenty-four hours 32·9 heat-units, and on slight work, 34·9 heat-units, so that a person weighing 70 kilos (10 stones) if lying in bed would require 2303 heat-units, and if going about 2443 heat-units; in addition he needs an allowance to compensate for the sugar he excretes unconsumed. The usually accepted heat-values of the ordinary elements of food are four heat-units for 1 gramme of albumin, the same for 1 gramme of carbohydrate, nine heat-units for 1 gramme of fat, and seven for 1 gramme of alcohol. As carbohydrates are necessarily excluded from a strict diabetic dietary the required values must be made up from albumin and fat with a little alcohol. Such a dietary, giving 2450 heat-units and containing not more than 500 grains of sugar-forming carbohydrates, is as follows:—

BREAKFAST

Fat bacon, 2 oz.; eggs, 2; tea or coffee, $\frac{1}{2}$ pint; cream, 1 oz.; Callard's bread, 3 oz.; butter, 1 oz.

LUNCHEON

Cooked meat, 3 oz.; green vegetables, 3 oz.; cheese, 2 oz.; Callard's bread, 2 oz.; butter, 1 oz.; claret, 4 oz., or whisky, 1 oz.

TEA

Tea, $\frac{1}{2}$ pint; cream, 1 oz.; Callard's cakes, 1 oz.

DINNER

Meat-soup or beef-tea, $\frac{1}{2}$ pint; cooked meat, 3 oz.; green vegetables, 3 oz.; cheese, 1 oz.; butter, 1 oz.; Callard's bread, 2 oz.; claret, 6 oz., or whisky, 1 oz.; coffee, 4 oz.

On this diet, in addition to the 500 grains of sugar derived from the sugar-forming carbohydrates, the patient's tissues receive a certain amount from the decomposition of albumin, roughly calculated as twice the quantity of urea excreted, so that if the patient is excreting 600 grains of urea we allow 1200 grains of sugar from the albumin of his food, which, with 500 from the carbohydrates, makes 1700 grains. How much of this is consumed in the body may be determined by estimating the quantity of sugar excreted in twenty-four hours, and subtracting it from the above amount. For example, a patient aged forty-two, weighing 126 lbs., required a diet containing 2205 heat-units, and was actually allowed one containing 2450 heat-units. His normal consumption of sugar was about 5000 grains. Upon the above diet, which contained no more than 500 grains of carbohydrates, he excreted 134 grains of sugar, and 792 grains of urea daily. He, therefore, consumed 1850 grains ($500 + (792 \times 2) = 2084 - 134 = 1850$), or about two-fifths of the normal amount.

It is desirable to continue the use of strict diet for some days or weeks in order that the tissues may regain part of their lost sugar-consuming power, and the duration of this regimen must depend upon the condition of the patient. If the sugar disappear completely, or almost completely, it need not be long, while if the sugar remain relatively high it must be persevered with for two or three weeks unless the patient shew signs of intolerance, *e.g.* by loss of weight, the presence of acetone or diacetic acid in the urine, etc. Whenever a change becomes desirable we should increase the carbohydrate food. It has been repeatedly pointed out that patients manifest individual differences which make it impossible to lay down hard and fast rules; but, speaking generally, such articles should be tried in something like the following order:—(1) milk, (2) potatoes and such vegetables and fruits as contain a low percentage of starch and sugar, (3) bread or toast, (4) malt liquors.

	Carbohydrates.	
Bread and bread substitutes—	Per cent.	Grains, per ounce.
Toast	73·00	319·0
White bread	63·00	275·3
Brown bread	49·25	215·0
Gluten bread	28·40	124·0
Almond bread	12·67	55·0
Callard's brown loaf	6·75	29·2
Protene bread	2·7	11·7
Vegetables—		
New potatoes	16·00	69·9
Old	20·00	97·40
Salsify	15·00	65·55
Ground artichokes	14·00	61·18
Green peas	12·00	52·44
Celery	11·80	51·5
Cabbage	11·63	50·6
Radishes	8·18	35·3
French beans	6·00	26·22
Cauliflower	4·55	19·6

	Carbohydrates.	
	Per cent.	Grains, per ounce.
Vegetables—		
Spinach	4·44	19·2
Asparagus	3·67	15·7
Lettuce	2·73	11·7
Endive	2·58	10·9
Cucumber	2·28	9·61
Fruits—		
Cherries	10·0	43·7
Apples	8·0	34·9
Pears	8·0	34·9
Ripe gooseberries	7·8	34·0
Green ,,	2·4	10·4
Red currants	6·8	29·7
Black ,,	5·0	21·8
Strawberries	5·0	21·8
Bilberries	5·0	21·8
Greengages	4·0	17·4
Apricots	4·0	21·8
Plums	4·0	17·4
Sour oranges	3·0	13·1
Cranberries	2·0	8·7
Milk—		
Condensed milk (without added sugar)	4·81	20·9
Cow's milk	4·81	20·9
Skimmed milk	4·75	20·5
Cream	3·5	15·2
Koumiss (English)	3·10	13·5

	Alcohol.	Sugar.	
	Per cent.	Per cent.	Grs. per pt.
Beer—			
Burton bitter beer	5·55	5·65	488·0
German lager beer	3·95	5·78	498·0
Harvey's sugar free	?	0·20	16·0
Wine—			
Moselle (still)	12·06	2·0	174·0
Hock	11·43	2·5	218·0
Bordeaux claret	9·40	2·3	220·0
Chablis	10·30	1·88	156·0
Spirits—			
Scotch whisky	49·40	0·00	
French brandy	55·00	0·00	

Milk is valuable, in the first place, because it contains a considerable amount of fat, and has therefore a high heat-giving power, so that it can replace some of the solid fat of the above-described dietary which is not always easily borne ; secondly, it contains lime salts, of which there is an abnormal loss in diabetes ; finally, the lactose or sugar of milk is more readily assimilated by most diabetics than many other forms of carbohydrates. The objection to the use of milk appears to rest upon an old observation of Bouchardat, who shewed by careful analyses made on a particular patient that the whole of the sugar of a litre of milk was excreted in his urine, and he therefore maintained that lactose was not assimilable by diabetics. This is a mistake ; many diabetics can take a pint of milk daily without any corresponding increase in their sugar

excretion. The quantity of starch contained in different vegetables can be seen by reference to the table. I desire to draw attention to the small percentage of starch contained in potatoes. This vegetable is almost universally eaten with pleasure, and 6 oz. while containing only from 350 to 600 grains of starch (the variation depending upon whether the potatoes are new or old), may usefully take the place of bread or bread substitutes at two meals in the day. While not losing sight of those individual differences already mentioned, potatoes should always be tried when it is desired to increase the amount of carbohydrates.

Fruit being an article of diet with which most people find it easy to dispense there is not the same craving for it; but reference to the table will shew how much can be allowed. It must be remembered that the sugar of fruit is mainly laevulose, which many diabetics assimilate when unable to consume dextrose. For this reason an apple, whether cooked or raw, can be eaten daily by many diabetics without any increased excretion of sugar.

The starch contained in wheaten bread in any form seems to be less well borne by diabetics than any of the carbohydrate articles already mentioned. Moreover, the percentage of starch is so much greater (being from three to four times that of potatoes, for example), that at first we must give some bread substitute made without wheaten flour, such as Callard's or Protene bread. Whenever a diabetic shews that he can assimilate a good deal of carbohydrate without any return of his former symptoms we may try small quantities of white bread, which should be preferably well toasted and not exceed 4½ oz. in twenty-four hours.

Von Noorden has recommended the use of oatmeal as being assimilated more readily than wheaten meal, but his results are not very convincing. "This oat cure consists in the daily administration of 200 to 250 grammes of oatmeal, best given in the form of gruel, every two hours; 200 to 300 grammes of butter, and often about 100 grammes of vegetable protein or a few eggs may be taken in addition. Otherwise nothing else is allowed except black coffee or tea, lemon-juice, good old wine, or a little brandy or whisky. Such a diet is often disliked by the patient, but he has always succeeded in getting over this difficulty. After three or four days of it, follow one or two vegetable days. Often even in this short time the purpose for which it was intended is found to have been attained; in other cases the same performance has to be repeated two or three times. It appears to be advisable to let a few days of restricted diet, or even one or two vegetable days, precede the oat cure; for when it immediately supervenes upon a mixed diet, the desired effect follows rather late."

It must be borne in mind that diabetic patients who are taking a diet tolerably rich in carbohydrates are not exempt from the risk of a return of hyperglycaemia. In order to diminish this danger the patient should abstain from all carbohydrate extras on one day in each week, while if at any time the sugar-consuming power shews signs of diminu-

tion or failure, strict diet should be again resumed for two or three weeks.

Unfortunately many articles, prepared and unprepared, but purporting to be free from starch and sugar, are not what they are represented to be; and every practitioner who undertakes the charge of a diabetic should make himself familiar with the simple means by which to detect the presence of these admixtures. In many instances this may be done by merely dropping a little weak iodine solution on the biscuit, bread, or flour. In a recent instance biscuits were sold as containing 2 per cent of starch, but which, on the addition of iodine, turned nearly black and proved on analysis to contain 46 per cent of this carbohydrate. The so-called gluten bread often contains nearly as much. But this test does not detect sugar, and the simplest means to detect the presence of carbohydrates is to boil the substance with dilute sulphuric acid, neutralise with caustic potash, and test with Fehling's solution. If all practitioners would protect their patients by doing this, or get this done before sanctioning any diabetic bread substitute, these fraudulent articles would be speedily driven out of the market. It is to be regretted that "Soy" flour should be recommended by some writers as free from carbohydrates, for it really contains about 24 per cent (Kinch),[1] while some Soy biscuits sold for the use of diabetics contain twice as much. Even almond flour and desiccated cocoa-nut may contain sugar, are sometimes adulterated by the addition of starch, and without analysis are not to be regarded as safe. The following directions for making cakes may be useful (90):—

Almond cakes: take ground almonds, 1 lb.; 4 eggs; two tablespoonfuls of milk; a pinch of salt (or saccharine); beat up the eggs, and stir in the almond flour; divide into cakes, and bake in a moderate oven for 45 minutes.

Cocoa-nut cakes: take finest desiccated cocoa-nut, $\frac{3}{4}$ lb.; ground almond, $\frac{3}{4}$ lb.; 6 eggs; $\frac{1}{2}$ teacupful of milk: mix and divide, and bake for 25 minutes. The addition of gluten flour makes lighter cakes, but involves the presence of some starch; it is also an improvement to ferment the dough.

In accordance with the principle of permitting all carbohydrates to be taken that do no harm, the observations of Külz, made as long ago as 1874, that diabetics could assimilate laevulose and inulin, have borne fruit in the production of commercial laevulose for diabetics. Two preparations are in the market—one a granular powder, made by Schering and Glätz of Berlin, the other a treacle-like substance, sold by Allen and Hanbury. Either of these may be ordered for diabetics in quantities up to $1\frac{1}{2}$ oz. daily, and are usually assimilated without causing any increased glycosuria. It is noteworthy that laevulose is not excreted as such, but appears to be converted into dextrose (glucose). Inulin, a

[1] The following analysis of Soy beans by Kinch is to be found in Frankland's *Agricultural Chemistry*, p. 198:—Water, 11·3; nitrogenous matter, 37·8; fat, 20·9; carbohydrates, 24·0; fibre, 2·2; ash, 3·8.

form of starch found in dahlia tubers, cannot be obtained commercially except as the tubers; but Dr. Hale White suggests that these may be cooked and eaten as a vegetable.

The use of alcoholic drinks is not necessary for any one in health, nor essential for persons suffering from diabetes; but a small quantity of alcohol helps to make up the deficiencies of a diet poor in carbohydrates, especially when the patient cannot take the large amount of fat required to compensate for this deficiency. Scotch and Irish whisky and Hollands gin are, as a rule, free from sugar. Still wines from the Rhine and Moselle districts, and light Bordeaux wines (vin ordinaire), contain very little; Burton bitter ale is nearly deprived of all sugar. A little whisky or Hollands, or one or two glasses of the above-mentioned light wines diluted with alkaline mineral water, may be allowed. Sugar-free champagne is advertised for sale. Malt liquors are superfluous and should only be allowed in exchange for equivalent quantities of other carbohydrates; a pint of bitter beer contains the equivalent of 488 grains of sugar; its effect on the urine should be watched with care.

Climate and Health Resorts.—There are, perhaps, many climates more enjoyable for an invalid than those of the British Islands, and there are several famous health resorts which offer special facilities for the treatment of diabetes; but diabetic patients do not bear well the fatigues and excitement of travelling, and it is the universal experience of physicians that too often they leave home to find a grave in a foreign land. No patient suffering acutely should be sent away from home, even for a comparatively short distance, if it can possibly be avoided; on the other hand, there is a large class of chronic patients who derive great benefit from change of climate or a course of treatment at one of the foreign spas. Speaking generally, a diabetic patient will do best in an equable, sunny, moderately dry, and not too hot climate; but as it is hardly possible to find these conditions in the same place all the year round, a change is needed according to the season. Moreover diabetics, like other people, have their idiosyncrasies, and these must not be lost sight of. Those who are braced up and strengthened by mountain air should not be sent to the sea, and conversely. The mineral water stations which have acquired a great reputation in the treatment of diabetes are Vichy, Contrexéville, Carlsbad, and Neuenahr.

The waters of Vichy are all of much the same composition, and contain bicarbonate of soda as their principal constituent. Their use is undoubtedly beneficial in diabetes, in which disease the alkalinity of the blood is diminished; there is some reason, indeed, as we have just seen, to believe that this diminution is the chief cause of diabetic coma. A bottle of Vichy water daily, taken with a little light wine or lemon-juice, is a prescription which is often useful, and one which can be followed in any part of the world. Vichy is a summer station, the season being from April to September; but as it is too hot in July and August, the best months to select for a visit are May, June, or September.

Contrexéville is situated in the Vosges, and is very suitable for gouty

diabetics in the autumn months; the water chiefly contains bicarbonate of calcium; its taste is pleasant, and it makes a very good table water. Carlsbad, in spite of its great vogue, probably owes its reputation less to the specific action of its waters than to the skill of the many eminent physicians who practise there during the season. Carlsbad water contains mainly sulphate of sodium, and is actively aperient. The journey thither is long and fatiguing, even when accomplished by easy stages and with all the comforts that money can ensure; the town, when reached, is beautifully situated, and in the early summer its surroundings offer many attractions to the visitor. The waters are decidedly useful if it be desired to overcome constipation or to treat obstinate stomach catarrh, or if diabetes be associated with marked obesity. The season lasts from 15th April to 15th September.

Neuenahr (Ahr valley, Rhine) is much more readily accessible from England than Carlsbad. Its waters contain sodium chloride, sodium carbonate, and carbonate of iron. The treatment of diabetes is carried out with much success at this quiet, pretty, semi-rural watering-place. The season, like that of Carlsbad, is from 15th April to 15th September; but July is better avoided. There are many waters which may be used by diabetics at home. Those of Vittel, Vals, La Bourboule, Royat, Giesshuebl, Apollinaris, Johannisquelle, and Bethesda may be employed, like those of Vichy or Contrexéville, as table waters. On the other hand, the waters of Marienbad, Rubinat, or Condal are aperient, and resemble Carlsbad water in that they depend for this property on the large proportion of sodium sulphate which they contain. Rubinat is especially useful to overcome the obstinate constipation which is so frequent and troublesome a complication of diabetes.

For the winter months, if it be desirable to send the patient away from home, the various watering-places on our own south coast, or in the Riviera, or Montreux, Arcachon, Biarritz, Algiers, Egypt, or the Canary Islands, offer in varying degrees the advantages of a warmer climate and change of scene; but in the majority of cases it is not necessary to send the patient away from home for the winter; as a rule diabetics do better when surrounded by the comforts of their own firesides.

Exercise and Massage.—I wish to lay especial stress upon the opinion that a diabetic should live as much as possible in the open air; and for this purpose walking, cycling, riding, driving, and such out-of-door games as golf should as far as possible be encouraged. At the same time we must warn our patients against the dangers of over-fatigue and catching cold. When indoors the rooms occupied must be well ventilated and plentifully supplied with pure air. The body must be suitably clothed, preferably in light woollen garments; and in this respect there has been of late years a great advance towards a proper perception of the rational in dress. Late hours, hot crowded gatherings, excitement, and, in severe cases, even games in which the danger of fatigue or over-exertion is lost sight of in the desire to win, must be strictly forbidden. When from any temporary cause the bodily weakness is too great to permit of active

exercise, massage or passive exercises may be usefully employed to promote nutrition and restore vigour to the muscles.

Baths and Bathing.—Bathing of all kinds may be allowed if it is not too prolonged, and does not involve violent exertion, as in swimming in a rough sea. The daily sponge-bath is not contra-indicated, and its temperature must depend upon the state of the patient. Warm baths and steam baths are much appreciated by those who have dry and irritable skins. The Turkish bath is useful in obesity, in which to some extent it fulfils the purpose of exercise.

Drugs.—Opium and its alkaloids possess a well-deserved reputation in the treatment of diabetes, and hold the front rank. They appear to have greater power than any other known remedy over the discharge of sugar from the liver; but this power is limited, and its extent is readily ascertained in each case. While it is easy to push the dose up to almost any amount, the results obtained do not support this practice, which is attended by the usual ill effects of the opium habit. Where we desire to diminish the total amount of urine secreted as rapidly as possible, a grain of extract of opium in pill may be ordered at bed-time; and it is permissible to give even more frequent doses of this remedy, or of morphine, for a time. But this should be done with discrimination, and the practitioner must not rely too much upon its influence. Codeia is a weak substitute for opium or morphine, and has little utility except as a placebo. Belladonna is sometimes combined with opium, and the combination acts well.

Salicylate of sodium has been credited with several cures (Sympson), but it is doubtful whether it possesses any specific action; the dose is from thirty to sixty grains daily.

Bromide of potassium exerts a favourable influence in soothing the nervous system, and is sometimes a valuable agent; the dose usually given is thirty grains daily.

Jambul, in the form of powder, extract, or tincture of the seeds of Syzygium jambolanum, is a drug which has great repute in the East Indies and Malayan Archipelago, but it has not justified this reputation in Europe.

Arsenic is a valuable tonic, and arsenite of bromine, or the liquor Clementis,[1] has been counted as a specific. Its value is at best doubtful.

The success of thyroid extract in the treatment of myxoedema gave a great impetus to therapeutic experiments with organic extracts in other diseases; and following the indication afforded by the recent observations on the influence of extirpation of the pancreas, numerous attempts to treat diabetes have been made by administering raw pancreas, or various

[1] Liq. Clementis, according to Häger, has the following composition: Acidi arseniosi. Potassii carbonatis āā gr. iss. Dissolve in five drops of distilled water in a test-tube, and warm until a clear solution is formed. Dilute with distilled water until the quantity weighs 153 grains. Then add 6·2 grains (4 drops) of bromine, and set aside for one day. The fluid is then ready for dispensing.

preparations derived from the gland, to diabetics. The result has been very disappointing. No good whatever has been effected by these means (H. W. G. Mackenzie). Even depancreatised dogs have not been benefited by it (Minkowski, Gley, and Thiroloix). Lépine has recommended hypodermic injections of pilocarpine to stimulate the pancreatic secretion, but with little success, if any. He has produced, however, from malt diastase, a glycolytic ferment which has been administered to several patients with good effect, the elimination of sugar being reduced in each case. He makes the ferment by macerating five grammes of malt diastase, obtained as pure as possible, in a litre of water acidulated with one gramme of sulphuric acid. After being allowed to digest for two or three hours it is neutralised with bicarbonate of soda, and this quantity (a litre) is administered daily. As it will not keep it must be made fresh every day. Numerous experiments have convinced me that the apparently good results of this and other similar ferments are merely due to the destruction of a certain amount of sugar in the stomach or intestine, so that as the patient absorbs less sugar he excretes a smaller quantity.

Karl Grube of Neuenahr has referred to the popular use of egg-shell powder in diabetes, and has seen general improvement without influence on the glycosuria follow the use of a teaspoonful daily of this remedy, or an artificial substitute consisting of carbonate of lime 93 parts, phosphate of lime and phosphate of magnesia 7 parts. He alludes to the great loss of lime in the form of earthy phosphate in the urine, and thinks the medication may tend to repair that waste.

Among other remedies recommended are Martineau's specific;[1] antipyrin in 15-grain doses thrice daily (Germain Sée); sulphide of calcium, $\frac{1}{4}$ to $\frac{1}{2}$ grain three or four times in twenty-four hours (Cauldwell); creasote, 4 to 10 drops daily (Valentine); phenacetin and exalgin (Dujardin-Beaumetz); camphor (Peyrand); iodoform (Moleschott); nitroglycerin (Kennedy); salicin, 30 to 270 grains daily (Dornblüth); carbonate of soda, $\frac{1}{2}$ to 2 oz. daily, with citric acid (Stadelmann); sulpho-carbolate of soda, 5 to 30 grains for a dose (Monckton): nitric or nitro-hydrochloric acid with tincture of nux vomica (Wilks); cocaine, $\frac{1}{4}$ grain three times a day (T. Oliver); pepsin, 10 grains three times daily (Gardner); ouabaïn, $\frac{1}{1000}$ gr. three or four times daily (Gemmell); phosphorus, gr. $\frac{1}{30}$ in perles, of which three to six are to be taken daily (Balmanno Squire); lactic acid (Cantani); hydrogen peroxide; uranium nitrate; liquid extract of ergot in half-drachm doses thrice daily (Dougall); benzosol, 60 to 75 grains daily (Piatkowski); strontium bromide, 30 grains thrice daily (Solis-Cohen); permanganate of potash and maté (Monin); piperazine (Hildebrandt).

[1] Martineau's specific:—Carbonate of lithium, 20 centigrammes (3 grains), added to a tablespoonful of the following solution: Arseniate of sodium, 0·20 centigramme ($\frac{1}{3}$ grain); distilled water, 500 centigrammes (80 minims); the mixture to be placed in an ordinary soda-water machine (gasogene), this quantity to be used daily with and between meals as a drink, alone or mixed with wine.

Dr. S. West has endeavoured to restore our confidence in uranium nitrate administered in doses up to ten grains three times a day. It was formerly used in doses of only one or two grains, and is apt to cause gastric irritation. A certain experience of the larger doses has assured me that even these have no constant or specific influence on the disease.

Treatment of Symptoms and Complications.—Neuralgia.—The sciatic and other pains often cease as the disease is controlled by general treatment, but the presence of these symptoms affords a justification for the use of opium or hypodermic injections of morphine. Quinine in 5-grain doses, or phenacetin or antipyrin in 15-grain doses may be tried. Butyl-chloral hydrate in 15-grain doses is particularly effective for facial neuralgia. The part may be painted with a liniment of equal parts of menthol (or camphor) and chloral, or the following ointment may be applied:—℞ Menthol, gr. xv., cocainae hydrochlor. gr. v., chloral hydratis, gr. iij., vaselini, ℥j.

Thirst.—This, too, is relieved as a rule by means which diminish the polyuria. It is useless to place restrictions on the patient's drink, but sucking pieces of lemon, or ice, or sipping very hot water, may diminish the demand, while hypodermic injections of pilocarpine stimulate the flow of saliva and relieve the dryness of mouth; glycerinum acidi carbolici in 5-minim doses is said to have the same effect. If it is desired to give lactic acid, this may be ordered as a refreshing drink in the form of lemonade, as follows:—℞ Acidi lactici ℥ss., tr. limonis ℥ss., saccharini solub. gr. iv., aquam ad Oj. Constipation in some cases appears to increase the thirst, and is almost always a cause of discomfort, if not of danger, from the absorption of the products of pancreatic digestion and intestinal fermentation. Any laxative which does not contain sugar may be employed, but the best remedies are the aperient mineral waters, especially those containing sulphate of sodium, of which Rubinat is the strongest and most effective. There are also the waters of Condal, Carlsbad, Marienbad; and of those containing sulphate of magnesium, Franz-Joseph, Hunyadi Janos, Friedrichshall, Aesculap, Apenta, and so on.

Oedema generally disappears rapidly after rest in bed. In obstinate cases massage and bandaging should be tried. Dickinson believes that the tincture of perchloride of iron in doses of 30 to 40 minims three or four times a day acts as a specific in this respect.

Pruritus.—This very troublesome affection in women can only be cured by great attention to cleanliness. After micturition the patient should always wipe the parts with a sponge wrung out of a saturated solution of borax; and for this purpose she should carry the sponge, when away from home, in a small sponge-bag under her skirts. It should be frequently washed in hot water, and wrung out of a disinfecting fluid. The parts, if sore, should be bathed with warm saturated solution of borax, then carefully dried, and powdered with boracic acid 1 part, French chalk 3 parts, or ointment of boracic acid, zinc, cocaine, or iodoform may be employed instead of the powder. Greasy applica-

tions may be more protective against the drip of the urine. The vulva should be washed twice a day at least with warm soap and water.

Sweating, where excessive, may be checked by the use of Dover's powder as a substitute for other forms of opium in the general treatment, and by the combination of opium and belladonna mentioned above.

Coma.—There is no remedy for this most serious complication. At the first indication of its onset the bowels should be got to act, if possible, by a brisk purge, or a large enema, and then an alkaline enema of half an ounce of bicarbonate of soda in half a pint of warm water should be allowed to flow slowly into the bowel and retained there. This may be repeated every hour. I have seen one case which I regarded as hopeless recover after alkaline enemas, but they have generally failed. In addition, citrate of potash, as recommended by Dr. E. S. Reynolds, in doses of half a drachm to a drachm, dissolved in copious draughts of water, may be given every hour; but neither these means nor the injection of normal salt solution, or of bicarbonate of soda (℥i.-℥ii. to the pint), into the veins, under the skin, or into the peritoneal cavity, nor inhalation of oxygen, nor hypodermic injection of strychnine, ether, or camphor, has in the long run proved trustworthy.

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R. S.

DIABETES INSIPIDUS

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Classification. — Under the names "Diabetes insipidus," "Diuresis," "Polyuria," "Polydipsia," writers have described a certain morbid condition of the system, characterised by excessive and persistent discharge of urine of low specific gravity which, save in exceptional cases, contains neither sugar nor albumin. Some authors apply any one of the above names to denote this urinary superflux, without reference to the quantitative relationship that may exist in individual cases between the urinary water and solids. Others, of whom Willis seems to have been the first, have attempted to form a classification on this basis. Thus, Willis divided cases of diabetes insipidus into three groups : (i.) those attended with an excessive discharge of aqueous urine, in which the solid matters are hardly affected—*hydruria* ; (ii.) those attended with a copious discharge of urine with a deficiency of urea—*anazoturia* ; (iii.) those in which the excessive discharge of urine was accompanied by a superabundance of urea—*azoturia.* Parkes also believed that diabetes insipidus is found under three different conditions : (i.) In cases pre-

senting no increase or decrease of tissue metamorphosis; (ii.) in cases with a decided decrease of tissue metamorphosis; (iii.) in cases with evidence of increased tissue metamorphosis, as shewn by the increase of some of the urinary solids. To this latter class of cases Parkes thought the term polyuria more apt than that of azoturia, for the latter only expresses the increase of urea; whereas in the cases quoted by him the fixed salts—chlorides, sulphates, and phosphates—were usually present in abnormal quantities. Again, the term *baruria* was introduced by Fuller to describe certain forms of dyspepsia associated with excess of urea in the urine, in which only the solid matter of the urine is increased, not the water. Lastly, Tessier recorded a series of cases closely resembling saccharine diabetes in the increased discharge of urine, the thirst, the neuralgic and rheumatic pains, the wasting, and secondary lung complications, but without any trace of sugar in the urine; a constant phenomenon in these cases was a very considerable increase in the excretion of phosphoric acid, whilst the urea was not proportionally increased, a point which Tessier thinks serves to distinguish them from cases of azoturia; this distinction he marks by naming the group *phosphatic diabetes.*

Though many writers still reserve the name diabetes insipidus for those cases in which a superflux of the urinary water is seen without any increase of the urinary solids, still it must be admitted that many cases present both characters. It therefore seems, so far as our present knowledge of these conditions is concerned, that this increased excretion of urinary water and of normal urinary solids respectively should be referred to two classes: (A) that in which the aqueous superflux is most marked —*hydruria*; (B) that in which the drain of one or more of the solid constituents of the urine is persistent—*polyuria*: this classification will be used in this article.

A. HYDRURIA.—**Definition.**—Hydruria is the form of "diabetes insipidus" characterised by a superabundant discharge of aqueous urine without any proportionate increase of the normal solids of the urine, and without the presence of such abnormal products as sugar or albumin, unless as a complication or towards the end of life.

Etiology.—Hydruria, according to hospital statistics, is a somewhat rare disease. Gerhardt states that 55 cases were found amongst 113,600 patients treated from 1877 to 1896 in the Berlin Charité, and Eichhorst saw 7 cases amongst 35,942 patients at Zürich. According to the register of the London Hospital from 1876 to 1895, only 8 cases were admitted during that period, of whom 4 were males and 4 females. The average age was 36 years; the youngest patient was 23, the oldest 55 years of age. The following table of 69 cases, collected by the late Dr. Ralfe, gives the number of cases in each decimal period, and of males and females:—

[TABLE

Age.	Males.	Females.	Total.
1 to 10 years . .	12	7	19
10 ,, 20 ,, . .	6	3	9
20 ,, 30 ,, . .	6	4	10
30 ,, 40 ,, . .	15	7	22
40 ,, 50 ,, . .	4	2	6
50 ,, 60 ,, . .	0	1	1
60 ,, 70 ,, . .	1	1	2
	44	25	69

It will be seen from the above that the two periods of life in which the disease is most frequent are those of early childhood and early middle age, and that the proportion of male to female cases is about two to one up to the age of 50; after that age the number of cases is too few for a fair estimation. This table accords with the view expressed by other authors, that the disease affects males in the proportion of "two or three to one," and that it is chiefly a disease of middle life; but it also shews that the disease is nearly as prevalent during the first decade of life. The youngest was a male child aet. 2, the oldest a fine, well-built farmer aged 69, who had always passed much more urine than he should.

Trousseau has remarked that it is not unusual to find patients suffering from "diabetes insipidus" whose parents were either glycosuric or albuminuric. This is especially the case with young children; but, on examining a considerable number of cases at all ages, I find that heredity has a wider range of constitutional dyscrasia than these two varieties. Thus, of twenty-seven patients whose family history I was able to trace, in twelve either one or both parents were tuberculous, and seven presented a distinct history of pulmonary tuberculosis. In five cases there was undoubted evidence of inherited syphilis, three of these were children; in three the parents had suffered from gout, two of these were adult patients; in two, one parent of each had died from heart disease with a history of rheumatism; two gave a family history of saccharine diabetes; in three, either the father or the mother had died of Bright's disease; but in at least two of these last cases the albuminuria may have been due to lardaceous disease consequent on phthisis. Futcher considers the cases may be divided into two groups, the idiopathic and the symptomatic, organic lesions being absent in the former and present in the latter, and that cerebral syphilis is the cause of many cases, and especially in the form of a syphilitic basic meningitis. In seven cases observed in the Johns Hopkins Hospital five were of the symptomatic variety, and four of these were of syphilitic origin.

Among the remoter causes among children malnutrition stands first and foremost. It is the children of the poorer districts of London

which furnish the bulk of the younger sufferers from this disorder, among whom such constitutional taints as tuberculosis or inherited syphilis are found, combined with ill-feeding and neglect. Certainly the disease is not common among children of the easier classes. Of thirty-four cases in which I have been able to investigate the personal history, eight were directly preceded by neglect, improper feeding, and a constitutional taint. Three cases seemed attributable to protracted worry and anxiety, four to alcoholism, three to acquired syphilis. In four cases the disorder appeared after influenza; three of these patients recovered. Three cases followed exposure to cold: one patient had fallen into the water, and sat a long time afterwards in wet clothes; another had drunk cold water when heated; and another had been much exposed to the great cold of the February of 1895. In one case the disease was associated with heart disease consequent on rheumatic fever. The affection may sometimes arise suddenly in the course of chronic heart disease; thus, I (J. R. B.) have seen it occur in a case of aortic disease whilst the patient was confined to bed under treatment. Three cases followed injury to the head; in these the diuresis after a time gradually declined. In three cases a tumour of the brain was found on autopsy. In two cases I found the disorder associated with aneurysm of the aorta, which is interesting in connexion with Dr. Dickinson's case of "diabetes insipidus" associated with an abdominal tumour invading the solar plexus.

To sum up the etiological factors, hydruria mainly affects early childhood and middle adult life; males suffer in the proportion of two to one of females; hereditary disposing factors mainly manifest themselves on the lines of tuberculosis, inherited syphilis, and gout; and among children the parents have often been either glycosuric or albuminuric. Of the more immediate causes anything tending to depress the nervous system may play its part; among such events are malnutrition and neglect, long-continued worry, heavy drinking, depressing illness such as influenza, exposure to cold, but it is probable that syphilis is a more important factor than any of these. Among the more direct interferences with the nervous system are especially basic meningitis, injuries to the head, tumours of the brain, pons, or medulla, and tumours pressing on the thoracic and abdominal nerve-ganglia, which probably agree in disturbing the vasomotor governance of the renal vessels.

Pathology.—The flow of urine can be greatly increased by the section of the renal nerves, also by puncture of the medulla, and hence the suggestion has been made that the pathology of diabetes insipidus is probably a want of control of the vasomotor renal nerves. The urinary flow can, however, be influenced in other ways; thus, the infundibular portion of the pituitary body yields on extraction a substance that not only has a marked effect on the blood-pressure, but is also a most powerful diuretic (Schäfer and Herring); this observation shews that such a condition as hydruria might be brought about by chemical as well as by nervous agencies. There are no characteristic changes in the

kidney. In cases of a long course changes in the renal texture are sometimes seen, but only such as are common to other diseases, and have had nothing to do with the urinary superflux. Dr. Saundby, however, thinks that a survey of recorded cases leads to the opinion that prolonged distension of the urinary passages by enormous quantities of urine during a number of years leads to a diseased state of these parts which cannot be regarded as a mere coincidence. The late Sir W. Roberts collected records of the post-mortem examination in fifteen cases, and in nine cases lesions of the kidney were present. In four of these atrophy and degeneration were marked, in one multiple abscesses were present, and in four the kidneys were enlarged and hyperaemic. It is interesting to trace the course of the nerves forming the renal plexus, as irritation from eccentric or distant sources may play a part in inhibiting the renal nerves. Thus, the nerves forming the renal plexus are derived chiefly from the solar plexus; as the right vagus and great and lesser splanchnics join the solar plexus, it is probable that branches of these nerves enter the kidney by way of the renal plexus. The splanchnics also send branches direct to the renal plexus; and the left vagus sends some fibres to the left kidney. They contain medullated and non-medullated fibres; and Krause has traced the latter as far as the apices of the papillae. Their mode of termination is unknown. Physiologically vaso-constrictor, vaso-dilator, and sensory fibres have been ascertained. The connexion through the vagus brings us into range with the medulla oblongata, and with many organs susceptible of tuberculous or syphilitic growths, or of sudden shock, such as chill. The solar plexus may propagate the effect of abdominal new growths or aneurysms. In long-standing cases the kidneys are mostly enlarged, especially about the cortex, to which may be added slight granulation of the surface if albumin were present in the later stages. Another common occurrence is dilatation of the pelvis, often so far as to resemble a cyst. Thus it has been suggested that there may be two forms of diabetes insipidus: one characterised by a normal or perhaps slight excess of urea, the other by a subnormal excretion of urea. The post-mortem evidence, however, is not yet sufficiently conclusive to decide this point. The diminution of urea may be due merely to the anorexia that accompanies the latter stages of the disease, whilst at the early period with a voracious appetite the urea is naturally increased. After a prolonged period of diuresis, it is easy to anticipate sacculation of the pelvis, which is a reservoir for the superfluity; since the patients, wearied by the frequent efforts at micturition, naturally try to retain their urine as long as possible. The cause of the great fall in the amount of urine, often to normal, on the accession of pyrexia is unexplained, unless it be by the immense loss of water that occurs in acute febrile states. The same effect is observed in diabetes mellitus; and I have noticed it in ascites, in which after the patient had been tapped twice the belly spontaneously emptied itself after a pyrexia (bronchopneumonia) lasting three weeks.

Symptoms.—The two symptoms of the hydruric form of diabetes

insipidus, which alone are characteristic of the disease, are a profusion of urine without sugar, of low specific gravity, and an intense thirst.

Urine.—In well-marked cases the urine may be increased tenfold—from two and a half pints to over twenty pints. The highest quantity I have myself observed was sixteen pints in the twenty-four hours. The diuresis may certainly be more extreme than in saccharine diabetes. Up to a certain stage of the malady the amount of urine passed is in excess of the total of fluids ingested, the excess being made up of the withdrawal of fluid from the tissues and from the food. Subsequently, as the appetite fails and the tissues become dehydrated, this disproportion is lost, and the total amount of urine falls below the aggregate of the fluids taken in. The colour of the urine is variously described as of a pale yellow or greenish tint. In characteristic cases, when fresh and examined through a tall glass cylinder, it has often a pale bluish tint, like that of the Sicilian aquamarine. When first passed it is faintly acid, or perhaps more frequently neutral; but it soon becomes turbid from the deposition of epithelial cells, and from ammoniacal decomposition causing precipitation of phosphates. The specific gravity is always low, but the statement that it has been known to fall as low as distilled water is founded on error of observation. The lowest specific gravity taken at 60° F. was, in a child, 1·002; in an adult, 1·003. Even figures so low as these, when the amount of diuresis is taken into account, shew an increase in the amount of urinary solids above the normal; such an increase, however, does not denote an increased metamorphosis of tissue, but rather a washing out of the tissues by the excessive drainage, and an excessive ingestion of food. At the most the solid matters are increased by a fourth to a third. As may naturally be supposed, uric acid is not deposited from such dilute urines; but it not infrequently appears when from any cause the diuresis is very greatly diminished. The most usual deposits are those of oxalates and calcium phosphates. Glucose, though no feature of the disease, may occasionally appear in small quantities; or it may appear towards the end of the case, when indeed it may merge into saccharine diabetes. Inosite or muscle sugar is not an infrequent constituent; in some cases I have observed it in considerable quantities. Albumin may be present in small quantities throughout, but it usually makes its appearance towards the end of the case as a part of the usual dyscrasia.

The intense *thirst* is as well-marked a feature as the diuresis, if not more so. Clinical records tell us of heroic potations. Trousseau has recorded the case of a patient who consumed fifty pints of fluid daily; his urinary excretion was about fifty-six pints. The same patient, on one occasion when the amount of drink had been restricted, had drunk the contents of his chamber-vessel! There is no doubt that the thirst in cases of "polydipsia" exceeds that of saccharine diabetes; moreover, at first the urinary excretion continues in excess of the amount ingested, shewing that both the tissues and the food are drawn upon to supply the aqueous drain: as the disease advances the balance becomes more equal, and as dehydration of the tissues increases more fluid is required to make

up for the body loss; thus after a time the fluid ingested is more than the urine excreted.

Among other symptoms noticed in the progress of the disorder are *disturbances of innervation.* In the early stages the patient often complains of severe and racking pains in the lumbar region shooting down into the thighs. Among the minor disturbances of innervation are the frequent tendency to headaches, mostly in the occipital region; spasmodic affections of the gastro-intestinal tract, such as choking sensations, hiccup, and irregular action of the bowels; itching of the skin and other disturbances of the sensory functions, and certain spasms of particular groups of muscles. Impotence is a marked symptom throughout. Amblyopia without any special lesion is frequent. Towards the end insomnia is usually very distressing; it often passes abruptly into coma, preceded or followed by convulsions. Special nerves are not paralysed unless there be some corresponding cerebral lesion; nor are ophthalmoscopic changes found, unless it be in connexion with some concurrent disease, such as a cerebral gumma. As in saccharine diabetes, the reflexes disappear early, though occasionally they persist for some considerable period.

Nutrition.—Digestion is usually well performed, and this in spite of the enormous quantity of food that is often ingested in the early stage of the disease. The appetite, so long as the patient maintains his general health, is often voracious; and when we remember the large quantity of water required to supply the drain from the body, this is not to be marvelled at. As digestion is often performed with regularity, this "bulimia" may give rise to no inconvenience; but in cases that run a prolonged course, the appetite flags, as the health fails, and is succeeded by a distaste for food, which increases until complete anorexia is reached. By this time the disorder has made considerable advance; the tongue has become red and glazed, the mouth and throat are covered with sticky mucus; the bowels, which before were obstinately constipated, now alternate between that condition and severe attacks of diarrhoea, which often become uncontrollable and hasten the patient to his end. Generally one of the troubles is a persistent flatulence which no plan of diet or medicament seems able to relieve. Emaciation sets in very early; but so long as the appetite is maintained it does not progress so rapidly as towards the later stage, when anorexia becomes a prominent symptom. Then the muscles rapidly waste, and the skin becomes wrinkled and assumes a dry, withered appearance. The extreme muscular weakness makes even the slightest exercise impossible, and the patient either takes to his bed or rests all day on chair or sofa.

The *temperature* is always subnormal, unless it be raised for a time by pyrexial affection; in this case it has been noticed that the abundant discharge of water falls considerably. The pulse is always feeble and easily compressible, with no sign of high pressure.

Among the symptoms that close the case, *simple exhaustion*, followed by *drowsiness* passing into *coma*, is perhaps the most frequent in chronic cases among adults; this exhaustion is often precipitated by a severe and

uncontrollable attack of diarrhœa. A form of low congestive pneumonia often carries off the patient already reduced by the excessive drain. In those who have a tuberculous diathesis pulmonary consumption sets in early; in young subjects convulsions and coma often announce the end.

Diagnosis. — In well-marked cases of "hydruria" there is little difficulty in recognising the disorder by the persistent discharge of large quantities of aqueous urine of low specific gravity, the intense thirst, and the progressive emaciation.

It is distinguished from saccharine diabetes by the absence of sugar from the urine and the low specific gravity. It certainly happens, however, in "diabetes insipidus" that small quantities of sugar appear in the urine; but in my experience it is always transitory, and the specific gravity is never high.

The copious discharges of urine of low specific gravity by hysterical patients may for a time mislead the physician. But the general character of the patient, the usually intermittent recurrence of these discharges, and their amenability to anti-hysteric remedies, are sufficient to protect us from a false interpretation of the symptoms.

The discharge of aqueous urine from large cysts in the kidney may be misleading at first. The intermittency of the discharge, and the absolute deficiency of solid matter, ought to prevent error after the first observation. In cystic disease of the young the urine is often of a pale, limpid character, of rather low specific gravity, usually albuminous, but not largely increased in amount. The detection of the enlarged cystic organs usually determines the diagnosis; otherwise the absolute decrease of solid matters, while the excess of the urinary water, if any, is moderate, will settle the matter. Imposture is not readily detected, as it is only necessary to add the requisite amount of water to obtain the characteristic urine; but close observation will detect the cheat: indeed the patient could not suffer from the supposed diuresis without experiencing ere long the horrors of the water torture of the Holy Inquisition.

Frequency of micturition may sometimes be taken at first sight for increased discharge of urine. In the incontinence of adults evidences of obstruction of the outflow and the character of the urine itself are sufficient for diagnosis. The nocturnal incontinence of children is easily recognised; but a difficulty may arise in those somewhat rare cases in which the incontinence is diurnal as well as nocturnal: even here, however, a slight attention to the facts of the case will soon determine its nature. The act is almost unconscious, the child wetting himself when in school or at play without seeming aware of any mishap; the urine is of usual, or sometimes of increased specific gravity, and careful observation will shew that the quantity passed in the twenty-four hours is not excessive.

Prognosis. — The progress of cases of diabetes insipidus is very variable—sometimes they run an acute course from beginning to end;

sometimes they persist for a number of years without any visible deterioration of health beyond a feeling of weakness and general malaise.

Attacks that set in acutely generally follow some sudden shock; such as blows on the head, sudden chilling of the body when hot, excess of alcohol, or worry and anxiety. After injury to the head the disease seldom becomes pronounced. In these cases glycosuria is generally noticed during the early part of the case, and after persisting for some time passes off, leaving the patient hydruric; this condition, however, seldom lasts more than two years, unless some secondary brain mischief have resulted from the injury. Sudden chilling of the body when heated has also a tendency to induce the disease in an acute form. As mentioned above, the disease may appear suddenly in an acute form in the course of chronic heart disease. When the disease arises suddenly it may also subside suddenly. Anxiety and overwork have not, perhaps, so immediate an effect, but still one sufficiently well-marked for the patients themselves to refer to it.

When the disease begins insidiously, and runs a chronic course, the factors concerned are those which gradually undermine innervation, and especially the inhibiting function of the renal vasomotor system. Such factors are chiefly the acquired syphilis of adults, the inherited disease in children, chronic alcoholism, general self-indulgence, and such constitutional diseases as tuberculosis, gout, and rheumatism. Such patients seem to drift imperceptibly into the "leaky" state, and rarely come under observation till a very profuse discharge is established. Among middle-aged adults of this class there is generally a history of antecedent genito-urinary trouble, such as nocturnal incontinence of urine in childhood and of spermatorrhoea at and after the age of puberty: as the quantity of water discharged increases, there is a gradual decline of virile power till it is quite lost. Should the disease run a chronic course no troublesome intercurrent symptoms, such as boils, carbuncles, or cataract, arise as in diabetes mellitus; nor unless the patient be distinctly tuberculous do pulmonary troubles set in early. When they do it is generally towards the end of the case, and are then chiefly of a chronic nature—a kind of dry, slowly-advancing pneumonia beginning at the apex, and gradually spreading to the other lobes of the lungs. More usually the end comes with symptoms shewing the consequences of the protracted dehydration on the nervous system, such as tremor, spasms, giddiness, and minor epileptiform manifestations. In protracted cases of hydruria the final stage is often painfully prolonged, unless cut short by an attack of congestive pneumonia, of uncontrollable diarrhoea, or of coma preceded by convulsions. From extreme muscular weakness the patient is unable to leave his bed, and lies coiled up in it, for the most part in an apathetic state. The skin becomes withered and covered with brawny scales, the tongue is red and glazed, and the breath horribly fetid. The ravenous appetite by this time has given way to an extreme disgust for food, and even the polydipsia is much diminished. The abdomen is distended by flatulence; the bowels are constipated and

the motions scybalous; this condition, however, not infrequently alternates with severe attacks of diarrhœa. The emaciated patient succumbs to one or other of the affections above named, and dies in the last stage of marasmus. Finally, as Dr. Saundby has repeatedly urged, death may be due to the gradual destruction of the kidney (*vide* p. 216). This secondary wasting of the kidneys in not a few cases leads to fatal uræmia. Diabetes insipidus then may run a long course with little danger to life, or even none; on the other hand, it is attended by grave dangers in more than one direction.

All cases of diabetes insipidus, however, do not end thus fatally; for though the drain of water may be excessive for many years, the general health may continue good and the digestive functions be performed with regularity. In such cases "hydruria," as Trousseau has remarked, constitutes an infirmity rather than a disease. Again, in established cases, acute exacerbations may arise and threaten life, and yet subside, leaving the patient in the same state as before. The disease may also set in acutely, and with great severity, and yet speedily subside under careful tending and rest; this was the case with a sailor admitted into the Seamen's Hospital, one of four, who had been exposed in an open boat for several days at sea, suffering great privations and anxiety. This man, when admitted, was passing considerable quantities of aqueous urine, which gradually diminished with rest in bed and full diet, without any special medical treatment: he was discharged perfectly recovered. Remissions of the disorder even in well-established cases are not infrequent.

In the prognosis of any individual case the points already mentioned must be borne in mind. In mild forms of the malady, that are an inconvenience rather than a disease, an abatement may be looked for under proper management; always remembering that such cases may become aggravated, especially if we have reason to suspect any latent tuberculous or syphilitic taint. Cases that set in acutely from some direct shock to the nervous system are often the most hopeful; while those, on the other hand, that begin insidiously and progress gradually, are the least amenable to treatment. When, however, the patient has once entered the downward stage—marked by anorexia, profound emaciation, and great muscular debility—the end is generally approaching; and, although there may be some lingering, when that stage is reached the victim rarely survives six months. In children, especially in those of tuberculous parents, the disease never runs a protracted course; from beginning to end the period rarely extends to two years, and is usually not more than a few months.

Treatment.—Many drugs have been recommended for this disorder, each being in turn regarded as more efficacious than the others. Among the first to be mentioned is valerian, which owes its reputation to the strong advocacy of Trousseau, who used it in heroic doses; in one case the quantity of 160 grains in the twenty-four hours was progressively increased up to an ounce. In large doses, but far less than these, valerian soon disorders the stomach, and creates such nausea that it cannot long

be effectively administered. When doses of half an ounce to an ounce of tincture of valerian are given four times a day in a wine-glass of water, with some spirit of chloroform and sal volatile to disguise the nauseating taste, a very encouraging result often follows. But the effect is soon lost, and any attempt to increase the dose fails on account of the nausea. Attempts to give the drug in the form of extract enclosed in capsules have the same disadvantage.

Asafetida has been used as an adjuvant, but it has no apparent influence on the diuresis, and only adds to the nauseating effect. On the other hand, 5 to 10 drops of tincture of cannabis indica seem to counteract nauseating effects, and to have a quieting effect on the patient. In a case in which the sleep was much disturbed by the frequent rising to micturate, the diuresis was diminished, and the insomnia consequently relieved, by a draught consisting of 6 drachms of tincture of valerian, 10 drops of tincture of cannabis indica, and 30 grains of bromide of ammonium, with sal volatile and spirit of chloroform in a wine-glass of water on going to bed; half this quantity was also taken twice during the day-time. For some time this mixture diminished the diuresis by almost one-half. As soon as the drug loses its influence, or the patient becomes intolerant of it, it should be stopped at once, and resumed again after an interval.

Ergot is another drug that has a good repute, and deservedly so; I have found that it effects a reduction in the amount of urine when other drugs have failed. Yet here again its power is limited; for if long continued or given in large doses, symptoms of ergotism appear, and its administration has to be stopped. Thus a farm lad, who attended for a considerable time as an out-patient at the London Hospital, was at first placed on 10-drop doses of liquid extract of ergot thrice daily in an iron mixture. The average quantity of urine at that time was seven pints, which steadily fell to a little over three pints. The drug was administered for many months, and the patient gained in weight and improved in general health, but eventually signs of ergotism appeared.

Nitro-glycerin has also been employed with good results. In one case, a male, aged forty, admitted as in-patient to the London Hospital, passing a large quantity of urine, and at first treated with valerian and other remedies without much benefit, was placed on nitro-glycerin; at that time he was passing 12 pints of urine daily; after four days the urine was reduced to $2\frac{1}{2}$ pints, and the patient was discharged.

These are the chief remedies that have been tried and recognised as useful; some may be more effectual in one form of the disorder, others in another. But no such distinctions can be made till the history of a considerable number of cases comes under review. We do not need so much an enumeration of indiscriminate instances in which one drug is useful, as the actual condition of the individual, especially as regards his family and personal history, and the reasons for the employment of a certain drug, and the probable causes of its success or failure.

Among other drugs that have been employed may be mentioned

opium and its preparations, strychnine, quinine, belladonna, nitrate of silver, salts of zinc, iron, and arsenic. These are rather useful adjuvants than primary remedies. Opium and its preparations, unlike their action in diabetes mellitus, have but slight effect. This we can readily understand, since opium in the former instance checks diuresis by checking the formation of sugar in the system, and by diminishing the amount of this diuretic body passing through the kidneys; thus the beneficial effect of opium in reducing the diuresis in the saccharine disease is nearly always proportional to the diminished quantity of sugar excreted. In diabetes insipidus no such reduction in the superflux is observed, though some diminution does to a certain extent occur, which is no doubt due rather to a check in the metamorphosis of tissue and in dehydration than to direct action on the renal circulation. The preparations of opium are very useful in advanced cases to relieve the insomnia, restlessness, and subjective nervous symptoms, and to check the advance of pulmonary symptoms. Quinine and strychnine are useful tonics—the latter being especially useful when there is much muscular weakness and convulsive twitching. Iron is always useful as an adjuvant, and, combined with ergot, much increases the efficiency of the latter drug. The more soluble preparations are the most suitable and best tolerated. In the latter stages of this disease it is called for to check the profound marasmus and anaemia; especially when these are attended with extreme muscular debility and passive oedema. The salts of silver and zinc by their action on the central nervous system have proved beneficial when convulsions, twitchings, or symptoms of an epileptiform character set in. The valerianate of zinc has in some cases a decided power of diminishing diuresis. Arsenic may always be relied on to improve the general condition in all stages of the disease. It relieves the excessive appetite in the early and the anorexia in the later stages: under its action the skin often loses its withered appearance, and the bowels become less constipated. When given in combination with some of the special agents already mentioned I have observed that it greatly increases their power.

In syphilitic or tuberculous subjects specific remedies must also be employed: for the former, mercurials and iodide of potassium, but with caution; for the latter, iodide of iron and cod-liver oil. In a case, recorded by Dr. Robertson, of diabetes insipidus dependent upon disease of the bulb, the only remedial measure that seemed to be of distinct use was voltaic electricity. The positive pole was applied to the back of the head and neck by a large electrode, and the negative, duly insulated to within a quarter of an inch of the point, was passed along the floor of the nostril till it rested on the cervical spine. The strength of the current was gradually increased from a half to five milliamperes, and its duration from one to five or six minutes at each application. This was continued every second or third day for seven weeks, when the patient felt so well that he left the hospital to begin work on December 15, 1888. The urine, on admission 260 ounces and upward per diem,

gradually diminished in quantity, the amount ranging from 78 to 88 ounces in the twenty-four hours during the last fortnight of his residence. The specific gravity had risen to 1010 or 1012. He was three times in hospital, and obtained the same relief by this means from the polyuria on each occasion, though the palsy due to the central disease persisted.

Symptoms arising from intercurrent diseases must be treated on general principles. The only incident which calls for attention here is that of constipation. This is caused by the dryness of the faeces from the drainage of water from the intestines, by the progressive weakness of the muscular walls, and by the excessive amount of food ingested. The accumulation occurs in the lower bowel, and impaction of faeces is apt to occur there unless care be taken to evacuate its contents. Irritant purgatives, especially those that affect the small intestines, should be avoided, as very slight irritation of the mucous membrane often causes severe diarrhoea of an aqueous character. Purgatives, when employed, should be those that act chiefly on the colon and rectum, such as colocynth and aloes combined with strychnine and belladonna. Resort may be had to enemas of simple water with a little castor oil added; or, if the obstruction be severe, a glycerin enema may be used. Flatulence, which is a very troublesome symptom, is not caused altogether by fermentative changes; the gas, which consists chiefly of nitrogen, may be the remnant of swallowed air, as is the "flatus" in the "passio hysterica." It often comes on suddenly, even after relief has only just previously been effected. A pill consisting of extract of valerian and asafetida, with extract of cannabis indica, rubbed up with oil of rue or cajuput, may give relief; to this a small quantity of some opiate may often be added with advantage. Massage over the abdomen should be employed daily, both for the purpose of prevention and of relief.

The *dietetic* as well as the hygienic treatment, though not so important in the insipid as in the saccharine form, still requires careful attention. First as to the quantity of fluid the patient should be allowed to consume in the twenty-four hours. In view of the almost insane craving ("hydromania") for fluid, and the enormous discharge of watery urine, the question has been considered whether the diuresis could be controlled by placing limits on the amount of fluid ingested. The experiment has been made, and in some cases the reduction in the amount of fluid has been carried to a cruel extreme. No doubt restriction of the amount of fluid is followed by a diminution of the diuresis, and up to a certain point restraint may be imposed; but it must be remembered that at this point the tissues are drawn upon, and their dehydration will increase. In one case recorded by Trousseau, in which extreme restriction was resorted to, although the amount of urine was considerably decreased, the patient found "his strength decreasing, his sight failing, and his body wasting." The plan of procedure I generally adopt, when a patient first comes under observation, is to ascertain the daily quantity of fluid he is in the habit of taking and the daily diuresis. If these be nearly balanced I reduce the amount of fluid by two pints daily; if this

be followed by a corresponding fall in the amount of urine passed, I make successive reductions of about one pint every third day in the amount of fluid taken. When no further reduction in the diuresis is effected, no further reduction in the amount of fluid ingested should be persisted in; otherwise the deficiency will be made up by an exhausting drain on the tissues. Thirst is greatly alleviated by allowing the patient to suck ice and by the use of acidulated drinks to which a little gum-arabic or glycerin is added. When the appetite fails it is a good rule to add some nutriment to the drink; thus, milk and water, cocoa and milk, and light mutton-broth may be taken. A tablespoonful of raw oatmeal stirred in a quart of water, and flavoured with a little lemon-juice and grated lemon-peel, forms a grateful drink. Tea and coffee increase the diuresis and should be avoided; the same objection applies to effervescing drinks.

With regard to the food there is not the objection to the use of carbohydrates which exists in saccharine diabetes, and they may be freely used; so may the fatty foods. With respect to protein food, so long as the patient can digest and assimilate it, it may be given; but when anorexia occurs, and the process of metabolism is more or less arrested, then there is danger of overloading the bowels with undigested protein material, of the absorption of toxic bodies, and of the formation in the tissues of toxins resulting from incomplete metabolism. Then the amount of protein food should be limited, and given in as soluble and assimilable a form as possible. To aid reduction, the food should be minced; and some preparation of pepsin may advantageously be given with it. It is safe to place such a restraint on the voracious appetite as is not immediately followed by loss of weight or decline in strength; otherwise, if the patient enjoy his food and digest it well, no great severity need be exercised. By interposing small meals between the chief ones of the day much of the craving may be allayed; thus, a plate of porridge, a basin of soup, or a poached egg is often sufficient to stay the stomach till the hour for the regular meal arrives. The amount of salt taken with food should be extremely small on account of its diuretic action; and of course all salted meats must be forbidden, unless bacon be permitted, and if so, none of the lean should be eaten, and care should be taken to select the fat parts (the back) from very mild-cured flitches. It has been noted that some patients with this disease are very tolerant of alcohol; others, on the other hand, are extremely susceptible to it. Except in moderate quantities alcohol certainly increases diuresis, and it should therefore be taken as a dietetic only, and then not in the form of spirits; a glass of bitter beer or some claret and water is quite sufficient. As the patient becomes weaker a glass of port wine twice a day may be added to this.

Should sugar appear during the progress of the disorder, the dietetic treatment will be based on the principles laid down for diabetes mellitus.

The *hygienic* treatment is of great importance. On account of the general relaxation of the nervous system, the patient should select a

bracing place of residence, near the sea if possible; if away from the sea, at a place of some elevation and with a dry subsoil. As the body-temperature is usually below the normal he should clothe warmly, wearing woollen both winter and summer; and he must avoid any sudden change of temperature. Exercise should be regular, but of a quiet character, and, above all, fatigue should be avoided. Walking, the gentle use of the bicycle, riding on horseback, and golf are the best forms. Rowing, football, or any exercise requiring sudden and violent movements, should be interdicted. In the later stages, when the patients, from muscular weakness, become unable to leave the house, efforts must be made to prevent their becoming bed-ridden. They should be made to rise daily, and encouraged to change their rooms, which, however, should be on the same floor. Massage to the limbs should be employed daily, but care must be taken not to overdo it. Care, too, must be taken of the skin, which from want of nutrition falls into a withered and atrophied state, and becomes extremely sensitive and painful to the touch, so much so that the patient cannot bear the weight of the bed-clothes; this intolerance adds to the insomnia and restlessness. The patient should be sponged daily with soap (carbolic) and water, and dried by the gentle friction of a soft Turkish towel.

B. Polyuria.—**Definition.**—Under the name polyuria three forms of urinary drain may be noticed: that of urea—"azoturia"; of the earthy phosphates—"phosphaturia," or, as Tessier and others name it, "phosphatic diabetes"; and "baruria," in which nearly all the solids of the urine seem to be increased together. To this form of urinary waste some continental writers have applied the name "demineralisation."

I. **Azoturia.**—This name was applied originally by Prout to a condition of urine characterised by the presence of urea in excess, though Prout did not distinguish between the increase of the urea and of the other solids. He pointed out, however, that it often preceded diabetes mellitus, but obtained little clue otherwise to its nature or pathology. Indeed, little advance has been made in this subject since his time. The affection is comparatively rare, and the cases are so chronic that they drift out of observation. No advance can be made until we distinguish between the single increase of urea, the increase of urea and phosphates together, and the total increase of all the urinary solids. I have met with many more instances of the so-called "phosphatic diabetes" than of pure azoturia.

Azoturia, strictly defined, consists in an increase of the elimination of urea out of all proportion to the other solids. As urea is a diuretic there is often in this state an increase in the amount of water; this increase must often mask the disorder, if the specific gravity alone be relied upon, and no precise determination of the urea made.

The disorder generally begins insidiously, with a feeling of languor; there is also an indescribable weariness, especially in the loins and thighs. The tongue is usually pasty and foul, and the edges of it red and

irritable. The stomach is evidently disordered, and intense cravings for food alternate with complete anorexia. If about this time the patient consult his medical adviser, and if his symptoms be properly interpreted, a little judicious management will probably prevent further mischief; and though the patient may still feel languid and tired, a visit to Carlsbad or Homburg, or better still, to some quiet grouse-shooting or deer-stalking resort in the Highlands of Scotland, will most likely put him right. If the opportunity be lost the disease will progress, the gastric disturbance will increase, and the tendency to anorexia and to thirst will become more pronounced. To the weariness in the loins succeeds actual pain, usually on both sides, though occasionally on one only; thus renal disease may be suspected, as I shall point out farther on. Boils often appear; not the large boils of diabetes mellitus, but small ones the size of a hempseed, with inflamed bases and yellow heads; these slowly develop, and frequently recrudesce. The skin becomes harsh, dry, and wrinkled from loss of adipose tissue, and the body-weight slowly but regularly declines. It is at this period that suspicion of diabetes mellitus arises, a more thorough examination of the urine is made, and the excess of urea and the absence of sugar are detected. Even in these extreme cases the patient may yet recover; otherwise the case may pass on, as Prout remarks, into diabetes mellitus, or the patient may perhaps die suddenly.

This disorder particularly affects male adults betweeen the ages of forty and fifty-five. Dr. Ralfe only met with one female patient, who was at the climacteric; it passed off as that epoch closed. Other writers on the subject mention the frequency of the malady among men. Of the pathology of the disorder we have no clear indication. It appears to be due to increased metamorphosis rather of tissue than of protein food. Although restriction of the latter has a certain effect in reducing the amount of urea the result is never considerable; and a purely carbohydrate diet has very little influence in restraining the amount of urea. The best diet is a moderate mixed one, all stimulating substances being excluded.

The *causes* of azoturia are obscure; all Dr. Ralfe's male cases were, with one exception, well-to-do men leading sedentary, busy, anxious lives, who for the most part lived neither wisely nor well. The other patient, an artisan, was evidently a man of constitutionally weak nervous organisation, and his trade was too heavy for him. The *prognosis* is favourable, especially in the early stages; even in advanced cases recovery will generally take place in two or three years unless diabetes or pulmonary complications ensue.

The *treatment* is to be directed to soothing the nervous system. The patient must rise late and go to bed early. The hours of work, if work be absolutely necessary, must be strictly limited; but if the patient can afford it and has strength enough for it, a cruise round the world is the best restorative. As an intermediate step a residence at Margate or a trip to the Highlands is to be advised. Of medicines I rely chiefly on

preparations of opium and hyoscyamus; the latter seems to have a specially soothing effect. A pill containing $\frac{1}{8}$ gr. morphine with 2 to 3 grains of extract of hyoscyamus may be given, and as a general tonic some mineral acid combined with a bitter infusion. The bowels should be kept well regulated. Every morning on rising the patient should have a "sea-salt" tepid shower-bath, the temperature of which is to be regulated in accordance with his power of resistance, and a good towelling must follow. Whilst care must be exercised not to let the temperature fall too low, it is important that the patient should not be relaxed by too hot rooms, or clothes which are too heavy.

II. **Phosphaturia.**—This name is applied to three conditions of phosphatic urine: (*a*) that in which there may not be any excess of phosphoric acid, but in which the earthy phosphates are deposited simply on account of the alkalinity of the urine, usually the result of dyspepsia; (*b*) that in which ammonio-magnesian phosphate (triple phosphate) is deposited consequent on ammoniacal fermentation in disease of the urinary passages; (*c*) that in which an actual excess of the earthy phosphates is formed, which, if the urine be alkaline, are deposited in dense masses in the urine glass, or if it be acid, give the urine considerable density.

To prevent confusion between these three forms Tessier has proposed the name "phosphatic diabetes." In normal urine the proportion of alkaline phosphate to earthy phosphate is as 2 to 1 grms.; yet in the cases we are discussing the proportion is not infrequently as 2 to 5 grms. Tessier gives cases presenting even higher proportions, such as 9 grammes. In these cases the urea is sometimes increased, but not always; therefore it is that Tessier contends that some name other than azoturia should be applied to this condition. Of my cases, taking one with the other, the urea was one-third in excess, whilst the phosphoric acid in combination with the earthy basis was trebled. In one case the urea was nearly doubled, whilst the phosphoric acid combined with the earths was four times the normal amount. In one case only was the urea normal, and in it the earthy phosphates were exactly quintupled.

The following table shews the results of six cases in which exact and repeated analyses were made:—

	Age.	Weight.	Quantity.	Sp. Gr.	Phos. Acid.	Urea.
		st. lb.				
Case 1[1] . .	16	8 7	2900 c.c.	1·010	5·2	51·0
,, 2[2] . .	24	9 0	9600 c.c.	1·004	6·0	...
,, 3[1] . .	25	9 0	2300 c.c.	1·015	7·8	33·5
,, 4[1] . .	27	8 7	1520 c.c.	1·022	5·2	41·2
,, 5[1] . .	37	12 7	2020 c.c.	1·018	7·6	48·3
,, 6[3] . .	20	9 0	3825 c.c.	1·016	6·7	87·2

[1] These four cases seem to approach most closely Tessier's description.
[2] This case passed rapidly into one of diabetes insipidus.
[3] This case became one of diabetes mellitus.

In all the cases, about eighteen in number, that have come under my observation, in which the phosphoric acid has been relatively in excess of the urea, very severe and distressing constitutional symptoms have been associated with the excess. These symptoms vary considerably, but they are all characterised more or less by great nervous irritability, derangements of digestion, great emaciation, and severe aching pains in the loins and back especially affecting the pelvic viscera. The urine is copious, of medium specific gravity, either acid and clear, or sometimes alkaline and whey-coloured from deposited phosphates. The termination of the disease is variable. Of the cases I have been able to trace six have died of phthisis, which in two was associated with pleurisy; three passed into diabetes mellitus; and one into rapid diabetes insipidus.

Treatment.—The main indications are rest and promotion of general nutrition. To attain this end opium or codeia should be given in full doses as soon as the patient first comes under observation. When, however, the nervous system is quieted, and the rheumatic and neuralgic pains are abated, the opiate should be discontinued lest it interfere with digestion. General tonics, such as iron, phosphorus, quinine, nux vomica, hydrochloric acid, and cod-liver oil, should be perseveringly used. If there be a history of syphilis, iodide of potassium should be combined with these remedies. Warm baths, followed by tepid douches, give great relief to the neuralgic pains, and also soothe the nervous system.

The soluble phosphates may be administered, but their utility in these cases is questionable. There appears to be no lack of these constituents in the system; the difficulty seems rather to lie in some want of power in the tissues to retain them.

The food should be light and nutritious, and milk one of its chief constituents. Alcohol should be avoided; even in small quantities it invariably increases the diuresis. The same may be said of coffee. The change to dry bracing air should be obtained if possible. The clothing should be warm, and the patient carefully guarded against cold, since in these cases a reduction of bodily temperature is always noted.

When in spite of these therapeutic and hygienic conditions the diuresis and excretion of phosphoric acid continue, though the general condition of the patient may temporarily improve, there is reason to fear that phthisis will supervene, or that the disease may assume the features of saccharine diabetes.

III. **Baruria** is the name applied by Fuller to certain urines which are characterised by a general increase throughout of their solid constituents, of one not more than of another, whilst the aqueous excretion remains tolerably constant. The specific gravity may run between 1·038 and 1·045, and at that it may remain for several days. If there were not an actual increase of the solid constituents one would attribute the heightened specific gravity to dyspepsia, or to a loss of water from some febrile condition; but in many cases I have found the urea, chlorides, and sulphates increased by one-half. This state seems to be brought about by a quickened tissue-metamorphosis, which may result in the formation

of more acid bodies in the proteolytic cells which may dissolve out the alkaline bases too rapidly. However, the cases I have met have been in heavily overworked persons. These patients have complained of much the same symptoms as the polyuric and phosphaturic classes, and have been relieved by the same treatment, especially by the frequent use of the sea-salt tepid douches. Arsenic also seems to be of special value in this "demineralisation." A medical man residing in the Midlands who suffered from this disorder drank freely of La Bourboule water, and said he could not do long without it.

CHARLES HENRY RALFE, 1897.
J. ROSE BRADFORD, 1907.

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J. R. B.

SEA-SICKNESS

By J. R. STOCKER, M.B.

SYNONYMS.—Fr. *Mal de mer*; Germ. *Seekrankheit*; Lat. *Nausea*.

Definition.—A functional disorder of the stomach often associated with giddiness, which affects travellers by sea, animals as well as man, and is chiefly felt by persons unaccustomed to its movements.

Etiology.—*Immediate Causes.*—The primary cause of sea-sickness is the irregular, unremitting, undulating motion of the vessel; or, in language more precise, the oscillation that occurs about its longitudinal and transverse axes. Of these two movements the pitching on the transverse axis is more unpleasant than the rolling on the longitudinal axis. We can and do anticipate the one, *i.e.* the roll, on account of its regular rhythmic character, and are prepared to meet it; the other, *i.e.* the pitching and tossing, is so sudden and irregular that, unless we have the gift, we cannot do so. But the motion is still worse when the two movements are combined in the tumult called a "wobble," and also when the vessel "scends": the uneasiness is felt principally at the moment of descent. The amount of suffering varies in direct proportion to the degree, the frequency, and the suddenness or quickness of the motion. Generally speaking, it is greater in a sailing boat than in one propelled with oars, and in a screw-propeller than a side-wheel steamer; it often

happens that one accustomed to the movement of a steamer, sailing ship, or row-boat becomes sea-sick when transferred from one into the other, the character of the motion being very different.

Much depends upon the weather and consequently on the season of the year; upon the situation one may occupy on board, the amplitude of motion being greater the farther one removes from the centre of the vessel; the direction of the long diameter of the bunk or berth, which should be always placed fore-and-aft and not athwartship; and upon the state of the atmosphere below: when the air is stuffy, close, and saturated with the smell of cooking, of paint or driers, of heated oil, of bilge-water, of vomit or other excrement, it is almost insupportable; and those who are inclined to be sea-sick much prefer to remain on deck. In ships which are or have been used as cattle-ships the odour of the manure, or of the disinfectants used in order to conceal it, is often very disagreeable. These nuisances, however, together with the disagreeable sounds of vomiting and retching, the thud, the occasional racing and vibration of the engines, the sight of swinging, swaying, oscillating objects, and the difficulty or impossibility of obtaining any rest even in the horizontal posture, can only be regarded as secondary or subsidiary causes of sea-sickness.

Reference should be made to the benefits derived from certain modifications of construction and design which have been introduced of late. These improvements consist mainly in the increase of the width of beam, the provision of bilge-keels, and the ballasting by means of water-tanks in the double bottom; all of these improve the trim of the vessel and make her movements far more easy. The substitution of the turbine for the reciprocating engine has reduced vibration to a minimum and prevented the racing of the engines, and will probably contribute as much to the strength and durability of the hull as it unquestionably does to the comfort and convenience of the passengers.

On the other hand the vomiting and sickness which affect old and seasoned sailors on board torpedo-boats appear to be due entirely to the quickness of the movement and to be caused by that molecular concussion to which Rosenbach attributes the malady itself.

Remote Causes.—The prime of life is the period most obnoxious to sea-sickness; infancy, childhood, and declining years are comparatively free; consumptive persons are seldom sea-sick. Women being less active, less accustomed to vicissitude, and less inured to travel, generally suffer more than men; at the climacteric period this proclivity is increased. The bilious temperament is more conducive to it than the phlegmatic, and the nervous than the sanguine; in respect of race and nationality the people of the northern parts of Europe suffer less than those belonging to the southern, the Celtic and the Scandinavian races less than the Teutonic, and the Teutonic than the Latin; the Americans are said to suffer more than we do. Townsfolk generally suffer more than country people; but an exception must be made in favour of those who dwell upon the sea-board, and those who take a pleasure in aquatic exercises. People who

pursue an active outdoor life suffer less than those who work indoors; keen and eager sportsmen suffer very little. Those of spare and frugal habits undoubtedly fare better than those addicted to the pleasures of the table; nothing favours an attack more than a debauch. Deaf-mutes and the insane are said to be exempt from sea-sickness; but so are many others who possess the soundest health of mind and body, and have no impairment of their senses. Imagination often plays an active part in its production.

Pathology.—Sea-sickness has been attributed to various causes: it is not my purpose to discuss them all, but the following list shews how materially they differ in character from each other:—

i. Concussion of the brain (Wollaston, Alderson, Neuhaus); ii. Anaemia of the brain (from heart failure in consequence of fear, Rawlins); iii. Hyperaemia or congestion of the brain and spinal cord (Chapman); iv. Functional impairment of innervation (Beard); v. Visual disturbances (Mayo, Wilson Fox, Graily Hewitt, and others); vi. Labyrinthine disturbances (Irwin, James of Harvard, and others); vii. Molecular concussion of the viscera (Rosenbach); viii. Concussion of the sympathetic ganglia or plexuses—solar, semilunar, coronary, or splanchnic; ix. Disturbance or succussion of contents of stomach (Glynn Whittle); x. Disturbance of equilibrium (Mayo and others); xi. Regurgitation of bile into the stomach; xii. Disagreeable odour from the bilges (Rawlinson).

It is unnecessary to deal with each of these hypotheses in detail, but assuming for the sake of argument that any one or all of those conditions may be present during an attack, they may severally be put aside upon the ground that they are insufficient to account for a disorder which betrays so many partialities, such obvious anomalies as this; or to explain the reason why so many persons are exempt, or how it is that practically all recover. Moreover, we should surely find now and then that some ulterior effect, some morbid consequence, some organic lesion would ensue, particularly in cases in which any local or constitutional proclivity to disease exists; in my own experience this has not happened, and if it did occur we should probably hear much more about it. As to the "visual disturbance" hypothesis Graily Hewitt, who was one of its warmest advocates and made a series of experiments with the object of determining the influence exerted by the sight of shifting images, came to the conclusion that although they added much to its discomfort they could not be held entirely responsible for producing the malady, and he admitted that the blind are not exempt. Driven from this untenable position, those who have been looking to the nerves of special sensation to supply them with a clue to the elucidation of the mystery have resorted to the ear—a clue, in my opinion, equally deceptive—and pursue a similar train of reasoning to that pursued by Beard, who argued that, since vomiting is one of the symptoms of concussion of the brain, it must in sea-sickness be due to a series of mild concussions of the brain. These authors, however, deal mainly with the vertigo and assert that, as vertigo is a prominent condition of sea-sickness and is often due to some affection

of the semicircular canals, the secret of sea-sickness is to be found in this locality; they attribute it either to some mechanical disturbance of the endolymph or to an irritative hyperaemia of the membrane, and recommend for its abatement the use of counter-irritants behind the ear. In support of this hypothesis James states that deaf-mutes are comparatively if not absolutely free from the complaint, and that they also suffer less when seated in untwisting swings. But even if it could be proved that the vertiginous effects are really due to these disturbances, it would be quite unwarrantable to conclude that sea-sickness itself is due to them. The truth appears to be that both the visual disturbances and the labyrinthine (if such there be) unite with the other uneasy, disagreeable sensations that occur on board ship in making matters worse, in adding to the discomfort of the sick, and in frequently precipitating an attack, but that they cannot be regarded as prime or essential factors in producing it.

The sense of sight, then, becomes an agent in the production of sea-sickness only by participating in the perception of the motion. The blind perceive it in other ways, but in respect of their blindness they are to that extent free from its influence. The difficulty of distinguishing the difference of certain flavours and odours in the dark, and even of knowing if a pipe is out or not, is well known, but this probably depends upon the want of practice. But there is no difficulty whatever in detecting the difference between deglutition and vomiting, advancing and retiring, rising and falling, or between inspiration and expiration, whether the eyes be shut or open, and it is probably in the latter way that the blind perceive it. There is no active motion here, and therefore the muscular sense cannot participate; the sensation is one that affects the respiratory apparatus only, either pleasantly or the reverse according to the direction and degree, the regularity or irregularity of the motion and the individual susceptibilities of the patient. The problem involved in the partial or total exemption claimed for deaf-mutes is probably capable of solution in a similar way.

The anaemic hypothesis has recently been resuscitated by Pflanz and also Binz. Pflanz, indeed, contrived a special sphygmograph for the purpose of taking the pulse at the ungual phalanx; but it would seem that if the brain does become anaemic, as they say it does and as the symptoms also indicate, it is quite as likely to be one of the effects as to be the cause of the disorder.

Let us now briefly refer to what Wollaston said upon the subject. He had observed that the mercury rose higher in the tube of a barometer when the ship began to fall; he concluded that the action of the blood upon the brain would be identical, and that this would cause a pressure or a blow upon the brain which by frequent repetition would result in vomiting and nausea. He experienced a necessity in his own case, and believed that others would perceive the same, to draw a deep breath at the moment of descent; this he attributed to an instinctive effort so to increase the capacity of his chest that the blood might flow more

freely into it, and thus relieve the pressure it would otherwise exert upon the brain; he found that the method gave him great relief, and he thought that this would prove to be the proper explanation and provide the proper remedy for sea-sickness. Alderson, who wrote in 1872, also expressed his firm conviction that this was the true explanation, and he argued strongly in its favour. But if it be correct the remedy would be obvious enough; the horizontal posture with the head lower, or, as Neuhaus puts it, to lie upon the side with the head bent upon the chest and the knees drawn up towards the chin, should be sufficient to arrest or to prevent it. The horizontal posture certainly does relieve it, but not so much nor so effectually as it should do if this explanation were correct; it may be partly true but it cannot be sufficient. Wollaston himself admits that he first perceived the nausea when waking up from sleep, but it evidently did not occur to him that the occurrence or continuance of sea-sickness when in this position is a strong if not insuperable argument to the contrary. Had Wollaston been acquainted with the researches subsequently made by Marshall Hall and others into the phenomena of reflex action, or carried his own investigations a little further, he would probably have found some cause to modify his views. Hall considered sea-sickness to be a reflex act. Deep inspiration taken at the moment of descent and constantly repeated will indeed provide the proper remedy, but principally, I apprehend, because it thwarts the tendency to the sudden and impromptu closure of the glottis which instinctively occurs in sea-sick persons. This spasmodic action of the glottis, either by direct or reflex action, at once excites the various conditions and sensations that immediately precede and accompany the vomiting, the choking being probably its direct precursor; and by constant repetition it soon induces the usual train of symptoms. Closure of the glottis as a voluntary or spontaneous action serves principally as an aid in making or increasing certain efforts; but whenever danger or discomfort threaten suddenly or alarmingly, as is commonly the case at the moment of descent, it occurs unconsciously, instinctively, and with great rapidity; and I believe that its constant repetition is the cause of all the trouble. It may be overcome by drugs, by habit, or by strong determination; or may happily be avoided altogether, as in the case of people who possess immunity. The difference, then, between a good sailor and a bad one appears to me to lie mainly in the difference in their mode of respiration; and the cause of the complaint seems to be seated neither in the stomach nor in the brain, but in the throat. Considering the very delicate sensibility of the larynx and the pharynx, their close proximity, their direct communication with each other, their connected innervation, the immediate contiguity if not the actual identity of the cerebral and probably also of the spinal centres for respiration and for vomiting, and finally the ease with which vomiting can be provoked by simple titillation of the fauces, we can readily understand how either momentary or constant irritation, annoyance, and distress of one may either by direct or reflex action be transmitted to and produce effects upon the other.

Symptoms.—The pleasurable sense of physical and mental exhilaration which is the first effect produced by a moderate degree of motion on the water soon gives way to a void or empty feeling at the stomach and a craving like that of hunger, but with no desire to partake of food. A feeling of oppression, weight, or sinking at the epigastrium; an increase of the mucus and saliva in the mouth and throat, which, instead of being swallowed by degrees, is gulped down; an occasional gape or yawn, sometimes followed or preceded by a sigh; a sudden pallor of the face; a little choking cough or hiccup; a hawking up and expectoration of viscid mucus; a sense of squeamishness, as though any moment vomiting might occur; flatulent or sour eructations; together with the general state of mind and body consequent thereon, namely, physical weakness, mental and nervous depression, giddiness and faintness, are sure and certain signs of a disordered stomach, and constitute the premonitory symptoms of an attack.

The principal symptoms, when an attack has once set in, may be divided for the purpose of examination into two groups: those belonging to the stomach and those belonging to the head. The first or gastric group comprises nausea, vomiting and retching, total loss of appetite, and constipation; the second or cephalic group, which the Germans call the "psychic," consists of faintness, giddiness, and headache. In some persons the gastric group predominates, in others the cephalic; but in the majority of cases both the stomach and the head participate alike, and comparatively few persons suffer from the one group to the exclusion of the other. I shall presently make further reference to this arbitrary distinction between the two, but must first describe the symptoms individually.

1. *Nausea.*—This was the name given by the ancients to denote sea-sickness; it is now used only to denote its leading symptom. Common enough in febrile complaints, in severe inflammatory diseases, in organic disease, in sudden and violent shocks to the system generally, and in divers forms of reflex irritation and excitement, this distressing feeling frequently constitutes the principal and sometimes the only symptom of sea-sickness. In susceptible people it may be provoked by the sight or even by the mere thought of undulating movements.

2. *Vomiting and Retching.*—Sea-sickness may begin and end in nausea, and nausea alone; but sometimes, and especially in the case of children, it consists of vomiting only, which may occur quite suddenly and unexpectedly, without any previous warning or indication, and without causing any inconvenience. But as a rule the vomiting is associated with nausea, headache, heaviness of the head, and giddiness, and is followed by most violent and distressing retching; the little nourishment that can be taken is immediately thrown up, but subsequently only the mucus and saliva that is swallowed, along with gastric fluid and green or yellow bile; later, pure bile comes up alone. Meanwhile the patient's strength begins to fail, he falls into a low despondent state, becomes indifferent alike to circumstance and fate, and feels inclined to wish (if wish he can) that death itself might end his sufferings.

3. *Loss of Appetite and Constipation.*—States of pleasure, we are told, are concomitant with an increase, and states of pain with an abatement of some or all of the vital powers; the same is true of comfort and distress. This will account in great measure, if not entirely, for the anorexia. Like the nausea of many other forms of gastric disorder, both functional and organic, the symptomatic nausea and anorexia of sea-sickness attain a quite exceptional degree, and are out of all proportion to the actual gravity of the case. Not only does the appetite for food continue in abeyance as long as the disorder lasts, but so also does that for every other kind of enjoyment or indulgence; even sleep, which often cannot be obtained until the vital powers are exhausted, is frequently disturbed and fails to renovate the failing powers of the system. In persons of a delicate, weak, and nervous constitution, who have only a slender stock of health and strength wherewith they may encounter such attacks, the recovery is often tedious and the effects are sometimes serious. Constipation, though partly due to lack of proper stimulation of the bowel by the gastro-biliary secretions, may partly be accounted for by the uneasy and distressing motion. In railway travelling the constant oscillation, vibration, and concussion to which the body is exposed, while most destructive to the plant, are more or less injurious to the animal system; not only are they most fatiguing to the body and trying to the eyesight, but they delay the process of digestion itself, and, by paralysing the natural peristalsis, induce constipation and sometimes end by causing sickness. It is said that these and other ill-effects of railway travelling have been considerably diminished upon certain lines by the introduction of the bogie car. So too in sea-sickness the motion causes constipation, and the constipation serves to augment the other symptoms and also in a measure to provoke them. The amount of urine also is diminished, but this depends more directly on the vomiting and the small amount of fluid swallowed.

4. *Giddiness and Headache.*—The headache is generally frontal, though sometimes also vertical or occipital; it is either of a dull and heavy, or of an aching, throbbing character, and is usually accompanied by a feeling of fatigue or soreness, and by aching or neuralgic pains at the back of the orbit. In the paroxysms of retching and of vomiting, the eyeballs are protruded and feel as though they were starting from their sockets, and the vessels of the head and neck are swollen. Each of these conditions may and often does occur in any ordinary attack of vomiting, and it would be needless to describe them here were it not that on account of their pertinacity and severity they become more distressing and pronounced; in part they may be due to eye-strain, but they are mainly due to the disorder of the stomach. The giddiness or vertigo, however, which is not to be regarded as an integral part of the complaint, but only as accessory, may possibly be due to visual or perhaps to labyrinthine perturbations.

Which of these two affections, the cerebral or the gastric, is primary, or whether they are independent of each other, is still a matter of

dispute. Probably there is a certain amount of truth in both of these conjectures, but few who contemplate the subject from a practical rather than a hypothetical or even an experimental point of view can doubt that, even if they are related to each other, the probability is very much in favour of the gastric rather than of the cerebral affection being primary. Giddiness indeed is often due to overloaded or disordered stomach, to gastric or to intestinal irritation; but in sea-sickness it is the symptom most readily relieved by lying down, and I therefore think it much more likely to be due, as Wollaston believed, to gravitation of the blood, combined, of course, with the sight of shifting images and disturbance of the equilibrium.

5. *General or Constitutional Condition.*—This has partly been explained in the statements made above. There are signs of shock or collapse of the nervous system, together with a quasi-febrile state; alternate heats and chills, with frequent chattering of the teeth and shivering, pervade the system; the eyes are dull and sometimes bloodshot from excessive straining; the countenance becomes extremely pale, inexpressive, and dejected; the complexion often has a greenish hue. The heart's action is depressed; the pulse and respiration quick and feeble; the skin cold and often clammy, while the tongue is moist but slightly coated. There is always some degree of hebetude; the mind is utterly indifferent to all around, no consecutive train of thought can be pursued, and the functions of the brain appear to be almost suspended. There is a general feeling of soreness all over the body, a sense of weariness and fatigue, and always more or less exhaustion or prostration.

Course and Progress.—If the voyage be a short one the symptoms usually subside as soon as it is over; nausea, giddiness, and headache may, and after a first voyage in particular often do, continue for a day or two, and sometimes even longer; but they are none the less on that account amenable to treatment. People sometimes have recourse repeatedly to short voyages or pleasure trips upon the coast in order to recover health; the rougher the weather and the sicker they become, the better are they pleased. Possibly in cases in which the liver is inactive and the digestive power feeble, or where the one is engorged and the other over-wrought, such voyaging may be beneficial; and it was probably on this account that Burton recommended it. In longer voyages four successive stages may be recognised, namely, those of depression, exhaustion, reaction, and convalescence; but the symptoms altogether rarely last for longer than a week, very often they remit in the course of three or four days, but recur if the weather become worse or the bowels costive, The worst time is in the morning, the patient almost always feels better towards the evening; but the reaction is gradual and progressive, and, with careful regulation of the diet and attention to the bowels, the relief experienced on getting accustomed to the motion of the vessel is permanent. In some few cases the symptoms never cease until the end of the voyage, even though it be a long and tedious one like that to India, China, or Australia; and we sometimes meet with persons, generally

women, who having come on board in feeble health or with a delicate constitution never leave their beds until they quit the vessel: on the other hand, such persons often find their health very much improved, if not entirely restored. In women, too, the menstrual molimina are usually more severe, the flow may either be augmented or diminished or sometimes interrupted altogether, and the period is frequently most irregular. In pregnant women, contrary to what might naturally be expected, miscarriage is not more prone to occur than it is ashore; but extra care should be always taken of them so as to avoid the risk of injury, or, if labour should occur, of post-partum haemorrhage. It has been stated by Barnes that a suitable pessary will often completely relieve an attack of sea-sickness in women who have some uterine displacement.

Complications.—Continued cases, such as those alluded to above, and others of a less protracted character, not infrequently assume *a low or adynamic form*, and require a considerable amount of care. The vomiting may abate somewhat, but nothing will remain upon the stomach, and ultimately the patient is quite unable to swallow any solid food and is averse from taking any liquid nourishment; he will only suck a little ice. The nausea is constant, the throat and mouth are parched and dry and the skin likewise, the temperature is above the normal, the pulse quick, small, and feeble, the respiration shallow and increased in frequency, the complexion sallow and the conjunctivae tinged with bile, the countenance expressive of anxiety, and a mild delirium, a very rare concomitant of sea-sickness, may supervene; in these cases there is always pain and tenderness of the abdomen, generally in the region of the stomach. These symptoms closely correspond with those of acute or subacute gastritis; and there is every reason to believe that in the act of vomiting the stomach may be wrenched or strained, or the mucous membrane irritated by the action of the gastric juice. Sea-sickness sometimes takes the form of diarrhoea. Jaundice is very rare except when the liver has been previously deranged.

It is not at all uncommon for *delirium tremens* to occur at sea, though whether this be promoted by sea-sickness may be considered doubtful; it is in my opinion much more likely to depend on constipation of the bowels. *Insanity*, particularly monomania of a suicidal character, is not infrequent, though it does not always follow that it depends immediately upon the motion any more than the delirium tremens does; or that it was occasioned by sea-sickness. Women who may or may not be hysterical are sometimes seized with fainting fits or fall into a state of frenzy in consequence of the violence and continuance of the sickness, or of the extreme weakness and prostration resulting from it. All such patients should be watched most carefully, and if necessary locked up.

Without denying altogether the possibility of *death* from sea-sickness pure and simple, we must hesitate before admitting its occurrence. I believe that it may occur in consequence of the gastritis already mentioned, in consequence of syncope, or perhaps from sheer exhaustion. In cases such as these, if death occurred, it might properly be attributed

to sea-sickness as the primary cause; but it would perforce be necessary to quote the secondary and more immediate cause also. This would apply still more particularly to cases in which death occurs in consequence of the entrance of a piece of undigested food into the glottis during vomiting—the immediate cause of death being of course not sea-sickness but suffocation. In all such cases it would be advisable to make a necropsy, and to look for some sudden cause of death such as apoplexy, aneurysm, a large, dilated, or fatty heart, aortic or mitral disease, pneumonia, ulcer of the stomach or bowel, strangulated hernia or granular kidneys; but in any circumstances it would still be difficult to regard a disorder, due to nothing more than a perverted function, as a principal or essential cause of death.

Sequels.—If the stomach has been overstrained by constant vomiting and retching, and especially when haemoptysis has occurred, patients should be cautioned not to partake of any rich or indigestible food, and may be advised that, if they are careful not to indulge their appetite too much at first, the symptoms will soon disappear. Fundamental impairment of health or constitution is very rare, and is probably confined entirely to such cases as have been dosed too freely with bromide or other sedatives, or to those whose sickness has been either caused or aggravated by improper feeding or injurious surroundings such as bilge air or bad odours from the cargo, neither of which, however, is likely to occur on board any first-rate passenger ship. Improved nutrition and freedom from worry and care are no small benefits, and there can be but few who do not gain thereby. The old Greek writers considered that sea-sickness was salutary provided it did not become too severe; and in my opinion it would probably promote the chances of conception.

Immunity.—Cases of complete exemption are not only much more numerous than is commonly supposed, but are also most instructive; for they furnish us with indomitable proof that sea-sickness cannot possibly be due to any change of an organic or physical description which would of necessity affect them as well as others, but must result from some functional disturbance to which the majority of people are liable but from which they themselves are free. Is their freedom due to some natural, acquired, or hereditary idiosyncrasy? Can there be anything peculiar in their diet or their habits which will account for it? Have they got the knack of voluntarily compressing their abdominal viscera and of so preventing it? *To do this they would probably have to draw their breath and close the glottis.* Or is it possible that they do not feel the motion, or that if they do they are able to ignore it? Lord Macaulay and Professor Sidgwick are said to have avoided sea-sickness by reciting poetry, and this no doubt diverted their attention from the sensations they would otherwise have felt. So, again, if one is occupied intently with any work or duty one may escape entirely, while those who have nothing to engage their attention may be lying prostrate.

Now that physical training has become so popular it should be quite an easy matter for active, energetic persons to acquire the faculty of

adapting themselves and their respiratory movements to the peculiar motions that belong to their new environment. For this purpose no exercises equal those of aquatic sports, such as rowing, swimming, and water polo, which are all so very suitable for the physical education of a maritime population, and have such a marvellous effect upon the development of the respiratory tract. To learn to breathe, says Checkley, is to learn the A B C of physical health, and his view is fully endorsed by the medical profession.

Treatment.—1. *Prophylactic.*—This consists mainly in careful regulation of the bowels and adjustment of the diet for a day or two at least, if possible for a week, before the beginning of the voyage. The diet should be of the lightest possible description consistent with the maintenance of health, and those who desire to avoid sea-sickness altogether cannot do better than adopt a purely vegetarian diet for the nonce; all alcoholic drinks should be forbidden, and nothing should be used either as a condiment, food, or drink which has the slightest tendency to confine or constipate the bowels; an antibilious pill or two should be taken, if necessary, every other night so as to bring the stomach and the liver into thorough working order. If the voyage has to be begun without much time for preparation, then one blue and two compound rhubarb pills should be taken the night but one before embarking; or, if greater expedition be required, a dose of Hunyadi or of Friedrichshall water, or some gentle saline aperient, such as Carlsbad or Glauber's salts, should be taken warm before breakfast. The traveller, however, should, never go on board upon an empty stomach; the last meal should be a hearty one and a glass of stout may be allowed. It has been asserted that the steady use of bromides for several days before the voyage will render people less susceptible to sea-sickness, and this I think may be admitted; on the other hand, the indiscriminate or continuous use of them must be unequivocally condemned. Hammond expostulated very strongly against this practice, and his experience is fully confirmed by many other medical men both ashore and afloat; De Vries employs only one or two doses of 20 grains each, with 2 or 3 grains of calomel, if necessary, before embarkation. The bromides all exert an anaesthetic action on the larynx and the pharynx; they were originally recommended for the use of travellers with the object of diminishing the excitability of the nervous system generally, but their chief utility, in my opinion, is due rather to their specific action on the throat, and, if my view of the pathology be correct, they would probably be quite as serviceable and at the same time less injurious were they in future to be used in the form of lozenges or pastilles; this might also possibly apply to certain other remedies to be referred to presently.

2. *Remedial.*—Atropine and hyoscyamine also act in some degree upon the throat, and are often used with great advantage; they may be taken in the form of sulphate once or twice a day, the dose of the atropia salt being from $\frac{1}{150}$ to $\frac{1}{100}$ of a grain, and that of the hyoscyamine salt from $\frac{1}{100}$ to $\frac{1}{80}$ of a grain. When the vomiting has set in the hypodermic

method may be employed with more success; Skinner used $\frac{1}{60}$ to $\frac{1}{30}$ gr. of strychnine sulphate, with $\frac{1}{150}$ gr. of the atropia salt for each insertion. This applies to cocaine also, and to the use of ergotin for menorrhagia.

Chloretone has been recommended in three doses of 5 grs. each, one to be taken every two hours, more particularly in cases of pregnancy. Dornblüth strongly advocates the use of an abdominal bandage, and the practice of deep inspiration.

Some deem it best merely to use a placebo, and leave the case in the hands of nature, the force of habit, and the recuperative powers of the system, while others have their nostrums. One man pins his faith on validol or valerianate of menthol, another finds chloral much more useful, a third prefers chlorobrom (a mixture of bromide and chloralamide); a fourth prescribes sulphonal, while a fifth asserts there is nothing like anaesthesine, which is usually administered in doses of 5 to 10 grs., but which Schliep gives in much larger doses, viz. 2 to 3 grammes (= 30 to 45 grs.). One would scarcely expect a remedy like quinine to be employed in this affection, but it has been found to be useful both by Semanas and Knapp, who regard sea-sickness as a marine malaria. Chance found that a dose of laudanum taken before he went on board made him absolutely indifferent not only to the motion of the vessel but also to the recollection and the dread of its effects, and he used it subsequently in smaller quantities with equal benefit.

One observer found that when he got his stomach firmly wedged between certain structures on the deck the sickness ceased entirely, but returned as often as he disengaged himself. Bonnet found some cases yield to hypnotic suggestion. Wolf employed hot compresses to the head. Madeuf gives a good account of continental opinions on the subject.

To lie flat upon the back with the head upon the level is undoubtedly the posture most approved by the public; it should be assumed before the vessel leaves her berth or moorings. Long preferred to let the head hang down a little; this position of extension is one that Howard recommended in cases of suspended animation, and, involving as it does a wider opening of the glottis, it would, I think, be more effectual. Mackenna thought it best to lie upon the [right] side and thus to keep the head below the axis of the body; the curvilinear attitude, recommended by Neuhaus shortly afterwards, has already been referred to. Chapman's ice-bag to the spine, though once a fashionable remedy, was condemned by Fordyce Barker and has fallen into desuetude, and the use of ice is now confined to sucking it or sipping ice-cold water or champagne in order to relieve the dryness of the mouth and throat and quench the thirst. Considerable relief may also be obtained by sucking lemons or, when these are all consumed, by using lime-juice, which when mixed with soda-water makes a pleasant drink. Beef-tea, beef or calf's-foot jelly, veal- or chicken-broth and a little gruel, arrowroot, or barley water often constitute the only nourishment that can be taken. Still it is always best to bite or at least to suck a crust

of bread, a piece of toast, a biscuit, or a rusk; while a sip or two of port, champagne, or even stout is often invaluable.

During a slight attack, or when the patient is recovering, condiments such as curry, Worcester sauce, and Cayenne pepper, though injurious in the worst stage, become very useful; while gin-cocktail or l'élixir végétal de Grande Chartreuse may be sometimes used with great advantage. To take a draught of cold sea-water, to bathe the hands and face in it, and to face the air on deck as much as possible, are simple measures of the greatest service.

Unless, then, it be to get over short voyages, the less we resort to drugs the better; they increase the nausea and oftentimes the vomiting, which is both discouraging to the patient and disappointing to the doctor; the warmest advocates of specific remedies candidly confess that they are of comparatively little use unless the treatment be begun before the voyage, a course to which very few who are bent either on business or on pleasure are willing to submit. Those who can resist the inclination to lie down and who keep continually moving, endeavouring to adapt themselves and especially the mechanism of their respiration to the motion of the vessel—which can best be done by means of calisthenics in the open air, by shouting or by singing—will soon become accustomed to it. If we use a drop or two of chloroform, a few grains of chloral, a little bismuth or magnesia, ingluvin or pepsin, or some peppermint or ginger; employ an occasional cathartic or some aperient saline and secure a daily movement of the bowels, if necessary by the use of an enema or suppository, we shall probably succeed in the majority of cases; others must be treated in accordance with the special features of their case; but of no bodily disorder can it be more truly said—"Our remedies oft in ourselves do lie."

J. R. Stocker.

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MOUNTAIN-SICKNESS

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Synonyms.—Fr. *Mal des montagnes*; Germ. *Bergkrankheit.*

Short Description.—Lassitude, nausea, quick and gasping respiration increased on the slightest effort, and headache, arising in all men at an altitude of 16,500 feet and upwards, and in some at lower levels. Vomiting, when it occurs, is probably due to fatigue or dyspepsia.

Causation.—The causes to which mountain-sickness has been attributed are fatigue, indigestion, diminished atmospheric pressure, heart failure, deprivation of oxygen, diminution of carbonic acid in the blood. Mosso, who puts forward the last conjecture, designates the condition by the not very apt title of "acapnia." If it must be admitted that the conditions are complex, it is becoming clear that the predominant cause is deprivation of oxygen. It is true that, even at lower levels, diminished barometrical pressure, fatigue, indigestion, and cardiac incapacity may contribute to produce states resembling mountain-sickness; it is true also that at higher levels it is not always or generally easy to distribute these factors in particular cases: nevertheless, by careful comparison of records and of physiological experiments, these additional factors are proved to be subordinate, variable, and contingent.

It would be difficult then and it is not of great importance, at any rate in this place, to apportion exactly the several causes of the confused effects of bad or imperfect training, of improper food, of casual indisposition, of lagging heart, and so forth, which may and often do appear in the course of mountain expeditions undertaken by more or less ill-trained persons; and

it is manifest that in these persons such disturbances will come on earlier and at lower levels than in those better fitted for the work; but these phenomena are inconstant, and many of them have no necessary connexion with mountains; on the other hand, the symptoms given in the short description are constant, and characteristic of all persons on high mountain or balloon ascents. The Jesuit traveller d'Acosta discriminated them with remarkable acumen.

Let us now shortly consider these alleged causes severally. For the supposition, many years ago, of *heart failure* as a cause of mountain-sickness I was chiefly responsible. That dilatation of the heart does occur in mountain-climbing has not been disproved by subsequent observers, rather the contrary; my observations on this subject have indeed been corroborated more or less by Conway, Roy, Leonard Hill, and others; but this change, if often coincident with mountain-sickness, is not the direct cause of it. Sir Martin Conway's admirable sphygmograms, taken at my suggestion on the Karakoram-Himalayas, have negatived an explanation of which indeed, on other grounds, I had become distrustful.

Fatigue is from the nature of the case a very difficult factor to eliminate; fatigue-products must frequently enter into the causation of particular cases of distress during high mountain excursions. Still, it did not avail Mr. Whymper that on one occasion he rode on horseback up to an elevation of 16,500 feet; mountain-sickness was not thereby averted. Again, mountain-sickness is mitigated instantly on remittance of effort, and aggravated as instantly by its resumption, perhaps even on so slight an effort as the adjustment of the screw of an instrument of observation; this, if not conclusive, strongly suggests some cause more immediate than the circulation of a poison in the blood. Metabolism is increased, it is true, on the High Alps, at any rate in the traveller if not in the native. This is not due to light nor cold, as it continues in persons at rest in a hut; nor does it take place at the seaside. On the other hand, if we hear much less of these mountain ailments than we used to do, it is in great part because of increased hotel comforts, upland huts, and a better knowledge of training. Furthermore, fatigue-products are formed and enter the blood in exercises other than mountain excursions, and have been so far studied by many observers that their effects are fairly well known, and can be distinguished from mountain-sickness in the strict sense in which I now use the name. Arctic explorers, great as is their labour, do not suffer from mountain-sickness proper; and the conditions of aeronauts are not altogether comparable with those of the mountaineers. Of the conservative effects of training I shall speak a little later, but the conjecture is no idle one that it may consist partly in the production of "anti-bodies." It is known that fatigued muscles contain a poison or poisons intensely poisonous to animals, but that animals can be made immune to them by small repeated dosage in the usual way. With fatigue vascular tone falls, the heart dilates, albumin may appear in the urine and the

temperature rises; whereas in training, as I shewed many years ago, hard exercise does not raise the oral temperature—or only by a tenth or two. The toxic cardiac dilatation thus induced must be distinguished from that which may ensue upon the high arterial pressure incident to extraordinary muscular efforts. In fatigue probably all the muscles are thus undergoing some destruction, and it has occurred to me that the languor which is often felt one or two days after a severe excursion may be the reaction after a toxic fever scarcely noted by the ardent mountaineer during the excitement of the climb. Guillemard and Moog, in five days on Mont Blanc, noted an abnormal formation of toxic alkaloids which, reacting on the kidney, diminished its water, azotised matters and salts; the blood held water in excess, the red cells spread peripherally, and the haemoglobin fell. The acuter symptoms were mitigated in a day or two by urinary flow, active formation of red corpuscles, and rise of haemoglobin (11A).

Indigestion, again, has been regarded as a potent cause of the malady we are now considering. And it is clear, from the experience of almost every member of the Alpine Club, that to mountain-climbers a healthy digestion is of fundamental importance. On a morning when his digestion is awry, the best of climbers may find himself, at no very high level, reduced to impotence by exhaustion, nausea, and even vomiting; on the other hand, great care in diet, a rigorous use of spare and simple food, may be as effective in postponing or preventing such distress. I well remember a retirement from an ascent of Monte Rosa on account of the repeated vomiting and prostration of a companion, one of the best climbers who ever trod the Alps, I myself being wholly free from the least discomfort; he was for the time dyspeptic, I was not, and mountain-climbing disagrees sadly with men who are out of condition. Partly because of my medical training, partly because I am never disposed to eat much when on the mountains, I have never suffered from this pseudo-mountain-sickness; thus, never having ascended higher than Mont Blanc, I was long disposed to disbelieve in any such malady: I regarded the state rather as a compound of fatigue, dyspepsia, and heart stress. The experience of climbers—such as Sir Martin Conway, Mr. Whymper, and others—at far higher altitudes is, however, conclusive that there is a definite disorder thus designated, one which not only presents uniform features of its own, but also appears with a remarkable uniformity in all persons, whatever their individual condition, at a certain altitude—about 16,500 feet, and in not a few 1000 or 2000 feet lower. Oddly enough, Mont Blanc seems especially favourable to mountain-sickness, more so, it would appear, than Monte Rosa at the same elevation: it is needless to say that the malady, or a modified form of it, may likewise come at a lower level if favoured in the individual case by idiosyncrasy or incidentally adverse conditions. As between one muscular man and another, climbing capacity (apart from that skill in the game which economises effort) depends upon strength of heart and depth of respiration.

A series of experiments on digestion at high altitudes is needed, especially on carbohydrate digestion. It is said that persons suffering from mountain-sickness pass diacetic acid in their urine; if so, this is due, I suppose, to the diminished oxygen tension in the atmosphere.

The next alleged cause to be discussed is the important one of *diminished barometrical pressure*. That such diminution is a potent cause of bodily distress and disease is well known in the so-called "caisson disease." But the symptoms of this affection present many differences from those of mountain-sickness. The factor in common between them is that of large changes of barometrical pressure. Now it is first to be noted that in caisson work the labourer is soon accustomed to the one pressure or the other; in fifteen or twenty minutes he is easy either at ordinary pressures on quitting the caisson, or in the high pressure within the machine. The danger lies in too rapid a transference from the higher to the lower pressure. In mountain work the transference is gradual; and the trying change is in the direction of falling pressure. A similarly graduated change from high to normal pressure is attended with no discomfort. But even the hardened mountaineer does not lose the true mountain-sickness by habituation. My old friend T. S. Kennedy, with Fischer, when shooting sheep in the Nepaul Himalayas, camped for some days at an average height of something more than 18,000 feet; and Kennedy told me that so long as they remained at and above this level, the disinclination for exertion and the rapidity of the breathing were never mitigated, save by night and repose. Mountain-sickness does not consist, then, in sudden transference from one pressure to another, but is a persistent disability.[1]

The electrical tension in the atmosphere is a factor that may have to be taken into account. The natives of Colorado recognise mountain-sickness under the term "Puna," and assert, with apparent truth, that the symptoms are much more notable and occur at a lower level when thunder-storms are brewing.

One more cause remains to be discussed, namely, *deprivation of oxygen*; this explanation, which we owe to Bert, was accepted by Roy in his study of Sir M. Conway's experience, and is also adopted by Dr. Hepburn in his careful and lucid discussion of the subject. It seems that this cause will probably suffice to explain all the essential phenomena of mountain-sickness, notwithstanding that so able an experimenter as Mosso takes precisely the opposite view. In attributing mountain-sickness to diminished CO_2 tension, Mosso must surely be wrong; and, if so, the source of the error may be explained by Aggazzotti's observation (p. 248).

Marcet demonstrated that fatigue under exertion is due as much

[1] In the inhabitants of lower levels that is; but Dr. Leonard Hill says that at Quito and Potosi girls dance half the night, and toreadors caper in the bull-ring. Moreover, he reminds us that condors circle five miles high round the peaks of the Andes in realms of cold attenuated air (Humboldt), while fishes swim in the profound depths of the ocean where the water pressure is two tons to the square inch. The poor physique and anaemic aspect of many highlanders is to be attributed to a low diet rather than to a low barometer.

to the exercise of the cerebral motor centres as to exercise of the corresponding muscles; and that the sense of fatigue, attributed too narrowly perhaps to the accumulation of fatigue-products, is due in part at any rate to the supply of oxygen being inadequate to meet the demands corresponding to effort. Mr. Glaisher's experience in a balloon, and the results of rebreathing expired air, support the belief that an imperfect supply of oxygen to the cerebral molecules, although it does not prevent the formation of a volition, yet takes away the power to carry the volition into effect. It is a matter of common experience with us all that muscular effort is more easily performed if a few long inspirations are previously taken; in this way the capacity for the effort can be increased. At 16,000 feet the oxygen per unit-volume of the respired air has fallen to a definite degree, to a degree apparently inconsistent with much activity, or even with strong volition. Moreover, there is a greater relative absorption of oxygen at pressures lower than 300 mm. of atmosphere; and, conversely, on surpassing 300 mm. the dissociation of oxygen is disproportionately increased. Zuntz and Schumberg at the Bétemps hut (9190 ft.) consumed respectively 37 and 25 per cent more oxygen than at sea-level, and at 12,500 ft. there was a rise of 54 per cent. Others in better training (Loewy and L. Zuntz) used only 10 to 20 per cent more; but the consumption does not sink nearer the normal than this. Roy, on consideration of the records, thought that with care a farther height than Conway's 23,500 feet would be attainable, and I think most authorities now agree that a greater height can be reached (cf. Workman, Graham, Fitzgerald). In man, when the oxygen of inspired air is experimentally reduced to 10 per cent, symptoms of dyspnoea begin; and, as Dr. Hepburn says, at this external percentage the oxygen in the alveoli must stand at less than 10 per cent. Now this fall of oxygen corresponds to 3000 mm. total atmospheric pressure, and to a height of 17,000 feet. By habit the limits of toleration may be extended somewhat; but perhaps the toleration rather than the capacity would be increased — the distress would be less perceived, although accurate observations might prove the functions to be more or less abnormal. In time, of course, the chest will enlarge in capacity, as indeed we observe in the inhabitants of highlands; yet Conway says that his coolies, who came from a region 10,000 feet in height, at 17,000 feet and upwards, were affected much as the rest of his party. Furthermore, a compensatory increase in the number of red corpuscles does take place, and probably requires a few days to become established (*vide* Vol. I. p. 666). The apparent increase during the first few hours is due to concentration of the blood by transudation of lymph out of the peripheral vessels, though, on the other hand, a higher velocity of current might even reduce oxidation. That there is an increase of haemoglobin per corpuscle cannot be asserted; though of course the total increase becomes considerable. Some readaptation comes about also by development of the respiratory neuro-muscular apparatus. It is said that shallow-breathing animals, such as rabbits, cannot live at heights of 18,000 feet for more

than two or three days, and that after death "fatty degeneration" of the internal organs is revealed; but this statement may have to be modified, for in the pneumatic-cabinet rabbits can support a diminished pressure equivalent to a much greater height if they are kept on a dry surface. A very remarkable result of Zuntz's experiments is the prodigious increase of O_2 consumption when climbing even at moderate elevations. Resting and fasting on Monte Rosa, the O_2 consumption per min. was 259·2 and CO_2 output 192·7; on resuming the climb the O_2 rose to 1329·0 and the CO_2 to 1017·0. In Berlin the figures were 232·8 and 182·7. Mosso's observations are to the same effect. The CO_2 in venous blood raises the O_2 tension by increasing the dissociation of oxyhaemoglobin, thus promoting supply to the tissues. Aggazzotti of Turin found an interesting proof of this reaction in that an addition of CO_2, in the proportion of 7 to 12 per cent, greatly aided the revivifying effects of O_2 inhalation. Dr. L. Hill's rule for climbers is that the respirations should not rise above 75 per cent, and after 15 minutes' rest should have fallen to 15 per cent plus. When the respiration rate exceeds 35 it gets shallower, the area of cardiac dulness enlarges, and venous pressures rise in the liver and other reservoirs.

Finally, a remarkable system of economies and readaptations is established by training, and by self-restraint and common sense can be maintained at its best, at any rate for many weeks. Townsmen may thus easily compass more than double the work, and in guides we may observe that the standard of ordinary men is even quadrupled. Besides the changes in blood and respirations already mentioned, no inconsiderable factor seems to be an economy in muscular movements; fewer muscles are used and antagonisms are moderated, so that the output of CO_2 for the same result may fall considerably, while by better osmosis the juices are quickened, water is dispelled, and fat is consumed. A curious illustration of the effect of derangement of muscular economy is seen on slight disablement, such as a twisted ankle or sore foot, whereby the walker's consumption of O_2 may be increased by 18 per cent.

That diminished pressure in itself, apart from the oxygen percentage, has any share in the production of mountain-sickness is an assertion often made, but at present without the support of definite evidence. Experiments in exhaust-chambers and in balloons do not correspond with mountain-climbing unless time enough be allowed for readjustment between the outside pressure and the pressure inside the body and its parts, a time which in the case of climbing is allowed. Andrew Smith, Leonard Hill, and other writers on caisson disease have shewn that the danger of this occupation lies in the want of careful provision for the gradual increase and reduction of pressures; and that with such precautions even extreme variations of pressure are borne with safety. Now the effect of reductions in oxygen-value, as pressure is gradually reduced, may be counteracted by securing a sufficient supply of the gas in the caisson or for the aeronaut. Devices for the provision of oxygen for climbers, divers, and aeronauts are on trial, and by such means no

doubt the highest mountains will be scaled. "Pneumatogen," for instance, is offered by a Vienna firm in portable tins each weighing 1 lb. Each tin is to give off O_2 for an hour, the only additional apparatus being an indiarubber breathing-bag. As Messrs. Mumm and Longstaff are taking a supply of it to the Himalayas we shall hear before long how it answers in practice. Mountain-sickness is felt more in sun than under cloud, and more in a trough, where the air feels stagnant, than in a wind. This effect of sun Roy attributes to a greater absorption of oxygen by melting snow; but, as Thomas found the sickness to begin at a lower level on rock than on snow, the explanation seems to be that by the heat the air is still more rarefied. A breeze rapidly diffuses the expired air, removing it from the neighbourhood of the mouth.

Although I no longer think that *heart failure* is the immediate cause of mountain-sickness, yet I am strongly of opinion that dilatation takes place to a greater extent in such exercises than is generally supposed; and in this opinion I am supported by Roy, and now by Dr. Hill. Into this subject I shall enter more fully in the section on "Overstrain of the Heart," in Vol. V.: meanwhile, I have only to point out that the establishment of any degree of dilatation of the heart must form part of a vicious circle in which the distress is multiplied. This tendency to dilatation is shewn in one or two of Conway's sphygmograms; but, as these were necessarily taken at rest, the immediate effects of exertion on the heart are scarcely to be found in them. To take tracings during exertion is impossible, they are untrustworthy enough as it is; but, as Roy suggested, observations might be made with a flexible stethoscope by a sufficiently skilled observer. The arterial pressures up to altitudes of 19,000 to 22,000 feet are said, during rest, to remain unchanged.

On descent it would seem that discomfort is felt down to a lower level than that of its onset (namely, to 13,000 feet); and this we should expect if at about 16,500 feet a greater relative absorption of oxygen has to be provided for. In some of Conway's sphygmograms the heart is slowed; this may point to vagus action sparing the heart; on the other hand, the ruling acceleration points probably in part to fatigue and certainly to the call of the tissues for more oxygen. Besides, the circulation of imperfectly oxygenated blood in the coronary arteries must account for some considerable part of the cardiac embarrassment.

Symptoms.—The symptoms of mountain-sickness are best described in the words of the sufferers. Mr. Whymper says: "When we arrived at 16,664 feet we ourselves were in good condition, which was to be expected, as we had ridden most of the way; but in half an hour I found myself lying on my back along with the Carrels, placed *hors de combat*, and incapable of making the least exertion. . . . We were feverish, had intense headache, and were unable to satisfy our desire for air, except by breathing with open mouths. This naturally parched the throat, and produced a craving for drink which we were unable to satisfy, partly from difficulty in obtaining it, and partly from trouble in sipping it. Before a mouthful was down we were obliged to breathe and gasp again

until our throats were as dry as ever. . . . We found it impossible to sustain life without every now and then giving spasmodic gulps, just like fishes when taken out of water." After some habituation Mr. Whymper says that the abiding symptoms were great lassitude and headache (and no doubt increased rate and volume of respiration which had become more automatic, or the bulb a little less sensitive to the venosity of the blood). Cheyne-Stokes breathing is commonly seen, at any rate during sleep, at no very extreme altitudes (I have often perceived it in myself); and is due, no doubt, to defective CO_2 stimulation. Mr. Clinton Dent experienced precisely the same symptoms, including "feverishness"; though he does not say, nor does Whymper, that the fever was measured by the thermometer. Now as to this symptom Sir Martin Conway's experience is decisive. That this man or that may feel and be "feverish" is probable, especially when we remember that fatigue-products are circulating in the system; but that fever is not an essential symptom of mountain-sickness is proved by the accurate thermometrical observations of Conway, who says: "Bruce's temperature and mine were both normal, notwithstanding that we plainly felt, and continued to feel, discomfort from the reduced atmospheric pressure." Mosso, in his experiments at the Regina Margherita hut, found that surface temperatures and mouth temperatures were subnormal, but *rectal* temperatures were increased. This was true of all his party. The relative behaviour of rectal temperatures in muscular work has yet to be fully ascertained and verified, but some researches have been made in control of the observations on phthisical patients. Drs. Burton-Fanning and Champion found rectal temperatures in the normal subject raised by exercise for a short time; for instance, three hours' bicycling produced a rise of 3·5° F. In a private letter Mr. Dent tells me that during a three weeks' residence at a height of 6000 feet (with Dr. Buckmaster and another friend, in 1896), the mean bodily temperatures of the members of the party were always below the normal. My own experiments (2), lasting over ten or twelve climbing days, shewed that the mean of the bodily temperature was not depressed though the curve was rather acuter in its course. On one day, indeed, I discovered in myself a sudden drop, like those recorded by Lortet, but with no sensation of discomfort or weariness. Probably in his case and in mine it was due to the impact of the cold air upon the cheek. In another passage of his narrative, Sir M. Conway says—on the summit of Pioneer Peak, 23,000 feet: "We ceased to pant for breath the moment the need for exertion was withdrawn, and a delicious lassitude and forgetfulness of past labour supervened upon our over-wrought frames. All felt weak and ill, like men just lifted from beds of sickness." These mountaineers, the Gurkhas excepted, did not suffer from nausea or vomiting, nor did any of the party from the haemorrhages which are recorded by Humboldt and others. Nose-bleeding and other strange mishaps which befell the first scalers of Mont Blanc are not perceived by their successors. It is a remarkable thing that many startling disorders

which beset the early explorers have vanished on a greater familiarity with mountains. Nevertheless, haemorrhages, such as nose-bleeding, gum-bleeding, and bloodshot eye, are not unlikely, of course, to occur on rapid diminution of atmospheric pressure, and therefore ought perhaps to be included in the list of symptoms which may attack mountaineers; in ordinary circumstances, however, they seem to be absent. Nausea and vomiting, whether due to fatigue, to poisons such as badly canned food, or to an aggravation of ordinary indigestion, would not in any case form part of the characteristic symptoms of mountain-sickness, as these effects would occur in like manner on excessive exertion at any level; yet it may be important to remember that Roy and Cobbett (in some unpublished experiments) found the digestive tract in asphyxia to be anaemic. Conway's two Gurkhas vomited at 17,000 feet and 22,000 feet respectively; but for caste reasons these men had been unable to modify their ordinary diet. Headache seems to be a constant symptom; and, as Dr. Hepburn says, may be explained on any hypothesis—cerebral anaemia, cerebral hyperaemia, etc.—as may be preferred by the individual commentator (12). Vertigo is mentioned, but not always, and with variable emphasis; perhaps it is of aural origin. One fine day, on the Lötschthaler Breithorn, we had to leave a healthy, well-trained comrade in a cave to await our return, so persistent and so distressing was the giddiness which attacked him somewhere about 11,000 feet. Palpitation also is mentioned by all observers, and is too familiar an experience to climbers to need description. I once took three friends up the Piz Morteratsch (12,316) without guides, and two of them, one an active and not particularly tired young lady, were so prostrate with palpitation, that I feared lest the uncovenanted service of a guide to carry his tourist up the last 100 feet should be my lot. But I found that if the patient lay down for ten minutes the heart soon fell to its proper rhythm, and by one or two repetitions of this device my friends got well to the summit. I suppose, therefore, the palpitation was rather a symptom of fatigue than of asphyxia, though aggravated no doubt even by so moderate a rarefaction of the air. Tinnitus, although mentioned by all climbers, even at relatively low levels, is but a result of unequal pressures upon the tympanum; however disagreeable, it is of secondary importance. The experience of the Schlagintweits confirms on the whole that of Whymper and of Conway. Dr. Hepburn thus sums up the proper symptoms of climbing at levels above 16,500 feet. *Acute*:—(*a*) increased respiration with spasmodic gulps; (*b*) incapability for exertion; (*c*) intense headache; (*d*) slight rise of temperature (rectal, but not oral, and such only as occurs during exercise at ordinary levels?—T. C. A.). *Chronic*:—(*a*) lassitude and fatigue; (*b*) increased respiration on the slightest exertion. The symptoms of the chronic group are those which continuously oppressed Mr. Kennedy and Fischer in Nepaul (private letters to myself). Warm-blooded animals from low levels seem to be affected in the same way. To these chronic symptoms I think coldness of the limbs may be added, and to

both classes palpitation of the heart and more or less vertigo; this last symptom may be due directly to diminished pressure, and if so, it probably belongs to the first or acute group.

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T. C. A.

DISEASES OF THE ALIMENTARY CANAL

GENERAL PHYSIOLOGY AND PATHOLOGY OF SECRETION
GENERAL PHYSIOLOGY AND PATHOLOGY OF DIGESTION

DISEASES OF THE MOUTH
DISEASES OF THE OESOPHAGUS
DYSPEPSIA
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GASTRITIS. GANGRENE AND CIRRHOSIS OF THE STOMACH
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GENERAL PHYSIOLOGY AND PATHOLOGY OF SECRETION

By J. Rose Bradford, M.D., F.R.S.

Secretion is one of the fundamental phenomena exhibited by living protoplasm. In secretion the cell protoplasm elaborates certain substances, and these are subsequently extruded from the cells. In the higher forms of life secretory processes are specialised so that the function of secretion becomes localised in certain cell-aggregates known as "glands." The substances elaborated by the activity of gland-cells are usually, but not always, extruded from the gland by solution or suspension in water; hence the discharge of water becomes the most obvious and one of the most important of the conditions of secretion. In a true secretion the gland elaborates the peculiar elements contained in the secretion; it does not simply remove them from the blood. The salivary cells make ptyalin and mucin, and the gastric cells pepsin; the kidney cells, on the other hand, simply remove urea from the blood: thus the elimination of urea by the kidney is not a true secretion, but rather an excretion. Other constituents of the urine, however, such as hippuric acid, are made by the renal cells. Some physiologists consider that a fundamental distinction between a secretion and an excretion is that the latter is passed to the exterior, without playing any further part in the economy; whereas the former, even if ultimately voided, fulfils some more or less important function meanwhile. A more vital distinction, however, is that previously mentioned, namely, that in a secretion the essential constituent is elaborated by the gland-cell; whereas in the excretion it is simply removed by the gland-cell from the blood or lymph circulating in the gland. Glands may, therefore, secrete or excrete; or again the same gland may do both. This double function is more especially seen in pathological conditions, when secretory glands not uncommonly excrete certain more or less toxic and abnormal constituents present in the blood-plasma. The biliary function of the liver is in part a secretion and in part an excretion; some of the characteristic constituents of the bile are elaborated by the liver itself, and other constituents of the bile, the cholesterin, for example, being, as it would seem, more or less effete products of metabolism, and of no further use to the economy, are thrown out.

Glands are generally provided with ducts by means of which the secretions or excretions are passed either to the organ where they are used, or to the outside. Certain glands, however, such as the thyroid, suprarenal, and pituitary, have no ducts; and the function of these ductless glands has always been a difficult problem. At one time their function was supposed to be the destruction of certain more or less toxic substances produced by the activity of the tissues, and this view is still held by some physiologists; by others it is held that these ductless glands also produce substances necessary either to certain tissues, or to the body at large, for the maintenance of physiological equilibrium. (The co-ordination of associated organs by these substances (hormones) is also dealt with in the article on "The General Pathology of Nutrition," Vol. I. p. 540.) This function has been called an "internal secretion," which denotes that the specific material elaborated by the gland is distributed to the tissues of the body, not by ducts but by the blood-vessels or lymphatics of the gland. It has been definitely proved that certain glands, such as the thyroid and suprarenals, elaborate peculiar substances necessary for the maintenance of life; these glands at any rate have internal secretions. Further, it is highly probable that many glands with external secretions have internal secretions also; the glycogenic and sugar-forming functions of the liver supply an instance in point. The formation of sugar in the liver, and the subsequent distribution of this sugar to the body through the hepatic veins, is really an illustration of an internal secretion. The material elaborated by the suprarenals is mainly of service to the vascular system; that produced by the thyroid is probably destined for the nervous system, and it would seem that the sugar produced by the liver is mainly required by the muscular system. Some physiologists do not consider the liver a sugar-forming, but rather a sugar-stopping organ. If this latter view were confirmed, the liver could no longer be regarded as having an internal secretion. Certain glands have no external, but an internal secretion only, for example, the thyroid, pituitary, and suprarenals. Other glands, such as the liver, the pancreas, and perhaps the kidneys, have both external and internal secretions. Finally, other glands, like the salivary glands, have external secretions only.

External Secretions.—The following processes occur during the complete act of external secretion:—(i.) The gradual elaboration and storage in the secreting cell of the specific organic constituents of the secretion, usually in the form of a zymogen; a zymogen is the antecedent of the ferment of the secretion—pepsinogen, trypsinogen, for example. (ii.) The rapid conversion of this zymogen into the actual substance found in the secretion—into mucin or pepsin, for example. This conversion occurs suddenly during the act of secretion. In some instances the conversion of the zymogen into the active ferment is effected in a more complex manner. Thus, the mucous membrane of the small intestine secretes a ferment enterokinase which acts upon the trypsinogen of the pancreatic juice and converts it into trypsin. Thus,

the pancreatic juice is inert until it reaches the small intestine (*vide infra*). (iii.) The sudden discharge of a considerable amount of fluid, the amount varying in the several glands; it is large in the case of the salivary and gastric glands, moderate in the case of the liver, small in the case of the pancreas. (iv.) The reconstruction of the protoplasm of the gland-cells, and not infrequently, as in the salivary and mammary glands, the replacement of used-up cells by freshly-growing and active ones.

Thus there is a slow elaboration of the specific constituents of the secretion, and a slow building up of the gland-cells anew, interrupted by a sudden discharge of fluid, together with an equally sudden conversion of the zymogen into the active and essential constituent of the secretion. In some cases the cells forming the gland actually break down into the constituents of the secretion; this is well seen in the mucous salivary glands and in the mammary glands. In other glands—as in the serous salivary glands, the gastric glands, or the pancreas—the zymogen is seen to accumulate in the form of granules, and after activity to remain only in the inner or lumen end of the cells. The formation of these granules can be shewn in certain cases to be under the influence of the nervous system. Stimulation of the cervical sympathetic causes these granules to be formed in abundance in the cells of the parotid, and the saliva subsequently secreted is abnormally rich in organic constituents. These granules are composed of a zymogen, and not of a ferment; this is shewn by the fact that a fresh extract made from a gland in full activity, such as the stomach or pancreas during digestion, contains no active ferment such as pepsin or trypsin. If the stomach be extracted after treatment with dilute hydrochloric acid, the extract is found to be powerfully proteolytic, and to contain an abundance of ferment. Extracts of fresh pancreas are possessed of but little digestive power, even when made from the pancreas of an animal killed during digestion; if, however, the pancreas be treated with oxidising agents the extract then obtained is a powerful digester of proteins.

The elimination of water by different glands during secretion is probably effected by various means. In the case of the salivary glands it is very abundant, and is closely dependent upon the nervous system, and especially on the activity of certain gland-nerves. In the kidney, on the other hand, the amount of water excreted is very closely dependent upon the rate of the circulation through the renal vessels, and there is as yet no proof of any nervous control of the excretion other than that of the vasomotor nerves which govern the renal circulation. In the cases of the stomach, intestines, and pancreas there is no clear evidence that the excitation of any nerve will cause the secretion to flow from these glands; although there is reason to suppose that their secretion is controlled in some way by the nervous system. The secretory activity of a gland is usually accompanied by a dilatation of its blood-vessels, and this is especially well seen in the salivary glands; yet even here this vascular dilatation is not an

invariable accompaniment of the secretory act, and there is abundant proof that the discharge of the water during secretion is not a filtration dependent upon the local vascular dilatation. There is no adequate explanation of the mechanism by means of which the salivary cell is enabled to secrete these large quantities of water as a result of nerve excitation. In certain glands the individual cells can be seen to swell up largely at the moment of excitation, and it may be that the cell-substance becomes suddenly hygroscopic, quickly imbibes fluid from the lymphatics, and as suddenly liberates it on the outward side.

Gland-nerves are concerned not only in the secretion of water, but, in the case of the salivary glands at any rate, they also contain fibres that exert an influence on the elaboration of the organic constituents of the secretion. Gland-nerves are therefore held to contain at least two sets of fibres; one set the so-called "secretory" fibres, most abundant in the cerebral nerves, such as the chorda tympani and nerve of Jacobson, the excitation of which causes the secretion of water; the other set the so-called "trophic" fibres, most abundant in the sympathetic, the excitation of which causes the gland-cell to elaborate the organic and probably also the inorganic constituents of the secretion. These two separate processes of secretion, the elimination of water and the formation of the specific constituents, occur not only at different times, but also, in the case of the salivary glands at any rate, under the control of different nerve-fibres.

Division of the cerebral nerves supplying the salivary glands is followed by a slow atrophy of the gland or glands, and the appearance of a slow, continuous secretion known as the "paralytic secretion." This continuous secretion is not produced by division of the sympathetic nerves; the cerebral nerve must be divided, whether the sympathetic nerve be divided or not is immaterial. A "paralytic secretion," following the division of the nerves distributed to the pancreas and intestine respectively, has also been described. In the case of the salivary glands the paralytic secretion is accompanied by an atrophy of the gland; hence it is probable that the reconstruction of the gland protoplasm is also under the control of the nervous system.

The nervous mechanism of secretion is most obvious in the case of the salivary glands (*vide* p. 272); in most of the other glands the evidence of this mechanism is as yet very imperfect. Dilute acids, if applied to the mucous membrane of the duodenum, are said likewise to cause a discharge of bile from the gall-bladder into the intestine. In the case of other glands, as of the stomach, it is probable that secretion can be produced reflexly, notwithstanding the absence of direct proof of the existence of secretory nerves (see p. 277). Secretion can also be produced by excitation of the higher portions of the nervous system; puncture of the medulla causes not only an increased urinary flow, but also a secretion of pancreatic juice; it is not certain that either is dependent solely upon the vascular effects produced by the puncture.

Further, the excitation of the cerebral cortex in the epileptic paroxysm, when produced experimentally, as for instance by absinthe, produces a notable flow of saliva.

Secretions are not only evoked by direct or indirect excitation of the nervous system, but may also be arrested or inhibited. The salivary secretion can be inhibited by fear, and the pancreatic secretion can be arrested by the excitation of the central end of an afferent nerve. Experimentally, the formation of urine by the kidney is sometimes entirely stopped by tying a cannula into the ureter; and, clinically, complete suppression is not uncommonly seen as a result of sundry injuries not directly implicating the urinary apparatus. It is difficult to believe that in these instances the total suppression of urine is due entirely to disturbance of the circulation.

The nervous regulation of the activity of the sweat-glands is of interest; although the sweat is practically an excretion, yet the nervous mechanism of these glands is as perfect as in the case of the salivary glands. The sweat-nerves leave the spinal cord by the anterior roots in the dorsal and upper lumbar regions, and then enter the sympathetic system to be distributed to the body at large in the sympathetic fibres. In some cases, as in the head, face, and neck, these nerve-fibres are found in the main sympathetic nerves of the part; in other cases, as in the limbs, the sweat-nerves are found in the nerves forming the limb plexuses; but these plexuses have received them by way of the communications existing between themselves and the sympathetic system. The sweat-nerves, although ultimately in the limb-nerves, do not leave the cervical or lumbar regions of the cord in the anterior roots of these nerves. These sweat-nerves have a very similar course to that of the vasomotor nerves, but they are quite separate and distinct from them. The excitation of a sweat-nerve causes a secretion of sweat, and after the division of such a nerve the area of skin supplied by it sweats no longer. Usually the secretion of sweat is brought about through the nervous system either reflexly, or by emotions, or by the chemical action of certain substances on the nerve-centres. External warmth does not cause sweating by any direct action on the glands, but indirectly through the nervous system.

Regulation of Secretion by Chemical Agency.—Recent observations have shewn that in some instances in which the mechanism of secretion would seem to be reflex and therefore nervous in character, the secretion is really produced quite differently and entirely by chemical agents. Thus, the passage of food into the duodenum has long been known to cause a flow of pancreatic juice, and this was regarded as a reflex act, but the real mechanism is as follows. The mucous membrane of the duodenum secretes a substance, "prosecretin," and this is converted by the acid present in the acid chyme into another substance, "secretin." This "secretin," when absorbed by the blood-vessels and carried to the pancreas, powerfully stimulates the secretory activities of this gland, and brings about the secretion of the pancreatic juice (Bayliss and Starling). In

the case of the stomach an extract of the pyloric mucous membrane, made with ·4 per cent hydrochloric acid, when injected into the circulation causes a secretion of gastric juice. Thus it is evident that, at any rate in the case of the digestive juices, chemical agents play an important part in bringing about secretory activity.

Uses of External Secretions and Excretions.—Excretions remove more or less harmful substances from the blood and tissues. They also eliminate water, and so help to maintain the amount of water present in the tissues at the normal standard. Thus indirectly they aid in the maintenance of the body temperature at its proper mean. Most external secretions are concerned in digestion either as lubricants, such as saliva, or as digestants, such as saliva, gastric and pancreatic juice; sometimes, in addition to this, secretions have some special action, such as the alleged antiseptic action of the hydrochloric acid of the gastric juice. The substances present in some excretions are removed, sometimes by what seems to be a roundabout channel; the urine, for instance, contains appreciable quantities of various aromatic bodies and conjugated acids that are apparently formed in the alimentary canal. It is quite possible, if the excretory activity of the kidney were greatly diminished, for these and other bodies, perhaps toxic in nature, to accumulate in the intestinal tract.

Some of the digestive juices are secreted in very large quantities; the secretion of gastric juice has been estimated at as much as seven litres a day: the great bulk of the fluid, however, is reabsorbed and not lost to the economy. The amount of fluid which daily passes into the alimentary canal and is again reabsorbed must be very large, and the daily quantities ingested and excreted form but a small fraction of the total amount. Of the solid constituents of these secretions also many are reabsorbed, and if, owing to a fistulous issue,—pancreatic or biliary, for example,—this be impossible, the percentage of solids in the secretion affected diminishes greatly; in the case of pancreatic or biliary fistula the percentage of solids may fall from an initial amount of 12 to 14 per cent to a final amount of 2 to 4 per cent. All the organic constituents, however, are not reabsorbed, since the urine contains small quantities of the various digestive ferments; moreover, there is a progressive destruction of ferments along the alimentary canal, the gastric juice destroys the activity of the saliva, and the pancreatic juice destroys the gastric.

Pathology of External Secretions.—In disease secretions are frequently affected, both in respect of the quantity of water eliminated, and of the nature and quantity of the essential constituents. The *salivary and buccal secretions* may be totally arrested, as in cases of xerostomia, so that great difficulty in swallowing arises from the mere lack of lubrication. A converse condition of excessive or of continuous secretion arises when there is some reflex irritation, especially of the mouth and tongue; as in cases of epithelioma of the tongue. Continuous dribbling of saliva occurs in bulbar palsy, and this condition may be due to the difficulty in

swallowing; yet in some cases the quantity of saliva seems excessive, and suggests a "paralytic secretion." Very rarely, and without the presence of any gross organic disease, copious and persistent salivation occurs, giving rise to a condition exactly opposite to that seen in xerostomia. The nature of these cases is obscure; they are not due to the taking of such drugs as the iodides, and they too seem to resemble the "paralytic secretion" of the physiologist.

The secretion of sweat is also profoundly affected in various diseased conditions; in febrile disorders, for instance, the secretion may be increased, diminished, or arrested. When increased, the increase is by no means always proportionate to the height of the fever. This is more especially true of phthisis, in which very copious sweating may occur with comparatively little fever, and with a pale anaemic skin. A direct contrast to this is seen in the dry and burning skin of pneumonia. The mechanism of the sweats in fever is not clear, but it is probable that they are dependent upon the excitation of the sweat centres and glands by abnormal substances present in the blood-stream, as well as upon the mere increased temperature. Heat causes sweating by its action as a stimulus to the nerve-centres. Copious perspiration is seen also as a result of pain, especially of reflex origin, as in biliary and renal colic. A peculiar instance of reflex excitation of sweating is that occasionally seen involving the face and neck, and occurring during mastication. Sweating confined to small areas often occurs as a result of nervous lesions, especially as a result of pressure on such a nerve as the cervical sympathetic, or from disease implicating the central grey matter of the spinal cord. In syringomyelia very copious sweating is often observed, and the transition from the area of copiously sweating skin to the adjacent dry area is often sudden. The sweating often coincides in its distribution with the analgesia so often seen in this disease. Conversely, the activity of the sweat-glands may be diminished in disease, as illustrated by the dry skin of diabetes and renal disease. The varying degrees of activity of the sweat-glands have a powerful effect in the regulation of the body temperature, since the skin and its sweat-glands form one of the channels by which variations in the loss of heat are regulated.

The Urinary Excretion.—Variations in the amount of urine excreted depend largely upon the amount of water lost by other channels, such as the skin, the lungs, and the alimentary canal; next to this the rate of flow through the renal vessels is the most important factor in determining the quantity of water excreted by the kidneys. In diabetes insipidus, on the one hand, and in the complete suppression seen after reflex irritation of various organs in disease, on the other, the nervous system must play an important part; but whether it acts directly on the renal cells, or only on the blood-vessels, is not definitely known. Chemical agents may also affect the quantity of the urinary excretion since the pituitary body has been shewn to contain a substance that has a very powerful diuretic action (20).

Calculi.—In glands provided with ducts, morbid processes often lead

to the formation of calculi, which consist usually of some of the more abundant constituents of the secretion. Occasionally the calculus-forming material is one, such as indigo, that normally is present in traces only; in some cases, indeed, as in that of cystine calculi, it is doubtful whether the substance be a normal constituent of the excretion at all. Purely inorganic stones are rare, but inorganic stones formed by accretion about an organic nucleus are not uncommon. The mechanism of calculus formation is sometimes obscure, but usually it is related to one or more of the following conditions:—

(i.) An excess of the petrifying constituent in the secretion. This mechanism is perhaps not so frequent a cause as was formerly supposed; but in many cases, no doubt, it holds an important place in the causation. Deposits of calcium oxalate occur not infrequently in the urine as a result of the ingestion of material containing considerable quantities of oxalate. Uric-acid concretions in the kidney depend sometimes, perhaps, upon an excess of the acid in the urine. Usually a mere excess of a particular ingredient normally excreted in a soluble form is not of itself sufficient to determine the formation of stone. Uric-acid concretions, indeed, may be found in the urine when the percentage of this acid is small and the urine dilute; on the other hand, large quantities of the acid may be excreted without the formation of stone.

(ii.) An alteration in the composition of the secretion so that the relative proportions of the various constituents are altered, and thus the chemical interactions dependent upon "mass action" are also altered. This is a most important cause of calculi, and the formation of uric-acid gravel often illustrates it. The uric acid in the urine is in the form of a soluble quadriurate, and for this salt to be formed it is necessary that a certain percentage of inorganic salts should be present. If this percentage be diminished uric acid may be precipitated as such, and a stone may be formed without the presence of any excess of uric acid in the excretion at any time. In the case of the biliary secretion an increased amount of calcium salts in the bile may lead to the formation of a biliary calculus, calcium forming an insoluble compound with the bile pigments.

(iii.) The size and shape of the crystals of certain salts are altered by the presence of colloidal substances in the secretion. Oxalates and other crystalline substances can be made to crystallise out from their solutions by the addition of some colloid matter such as albumin. The substance in solution not only crystallises out, but the crystals are frequently very large, and in this way may act as a nucleus for subsequent crystalline accretion. The exudation of blood or of some albuminous material into the renal pelvis may perhaps determine the formation of a stone in this way, and may serve to explain the occurrence of stones in one kidney only; although the calculus-forming material is probably present to an equal amount in the blood distributed to both kidneys.

Crystals of uric acid are affected profoundly in their size and shape by certain pigments in the urine, and thus variations in the amount of urinary pigment may modify the form in which the uric acid is excreted.

(iv.) It is possible that colloid material, such as nucleo-albumins, may act as a nucleus for the formation of stones. This action is distinct from the preceding, in that it is simply mechanical, whereas the essence of that described in the third section lies in the fact that the crystalline form is altered, at any rate as regards the size of the individual crystals. Gall-stones are sometimes formed long after an attack of catarrhal jaundice, and apparently as a sequel of it. It is possible that in these cases a cholangitis has extended to the gall-bladder, and that the exudation from the mucous membrane of the gall-bladder has determined the precipitation and crystallisation of the calculus-forming substance. In rare instances foreign bodies have been found as the nuclei of stones, but even in these cases it is possible that the foreign bodies produce the effects by means of inflammatory changes in the mucous membrane. Micro-organisms have also been found in the centre of gall-stones, but whether the calculi are to be attributed directly to their presence or to the chemical and physical changes produced by them is perhaps doubtful.

Diseases of glands resulting from partial or complete obstruction of the ducts are common. The duct of a gland is liable to become obstructed as a result of a mere alteration in the consistency of the secretion; from the presence of calculi; from the results of inflammatory changes in the walls of the duct, or from the pressure produced by growths in the duct or in adjoining structures. When the duct of a gland is obstructed the secretion does not cease at first; the usual effect is that the gland-ducts, and later the gland itself, become distended with a more or less abnormal secretion. Some of the constituents of the secretion pass into the circulation, either directly through the vessels of the gland or indirectly through the lymphatics. In many cases of long-continued obstruction the effects are complicated by the occurrence of inflammation of the walls of the duct. The ducts become greatly distended with more or less stagnant secretion, and in these circumstances an ascending microbic infection is liable to occur. This is especially seen in the case of obstruction of the bile-ducts. In the salivary glands, pancreas, and kidneys the secretion goes on at a sufficient pressure to distend these organs forcibly to a considerable size. In the liver the bile is secreted normally under a low pressure; thus jaundice results even when the obstruction to the biliary flow is slight, the stagnant bile passing into the lymphatics and veins of the liver. When a small calculus obstructs the exit of bile from the biliary papilla, but leaves the orifice of Wirsung's duct patent, bile may be driven by the contractions of the gall-bladder into the pancreas, and may set up acute haemorrhagic pancreatitis (Halsted and Opie). It has been shewn by Flexner that the bile salts are responsible for this effect, and that their influence is counteracted by the colloid constituents of the bile.

Toxic Substances.—Secretions in disease frequently contain abnormal and more or less toxic substances, and thus act as the means of their removal. In uraemia, the bile, the gastric secretion, and the sweat contain appreciable quantities of urea and other extractive bodies. In

cases of haemoglobinuria, due to the presence of free haemoglobin in the blood-plasma, the haemoglobin is excreted not only in the urine but in the bile also. These abnormal bodies are not taken up indiscriminately by all glands. Bile pigments, in cases of jaundice, appear readily in the urine; but even in cases of complete and persistent obstructive jaundice bile salts appear in the urine in but small quantities. The different secretions exert a more or less selective action upon the abnormal constituents removed by them from the blood-stream.

Microbic poisons are frequently removed from the body in secretions, more especially in the urine; if microbes are present in the general blood-stream the urine may contain them also. It is probable that many pathological processes, such as gastritis or nephritis, are dependent upon the removal by the stomach or kidneys of toxins and microbes from the general circulation. However, all disease poisons present in the blood are not necessarily present in glandular secretions.

Internal Secretions.—In the case of internal secretions the gland elaborates some substance or substances that are required to maintain the metabolic activity either of the tissues generally or of some particular tissue. The existence of internal secretions is most easily established in the case of glands without ducts, such as the thyroid and suprarenals; the difficulty is greater in cases like the liver, pancreas, and kidneys: yet even in these cases there is some evidence of the existence of internal secretions. The evidence on which the existence of internal secretions is based in the case of ductless glands, such as the thyroid, is as follows:—The complete removal of the gland is followed by death, and this event cannot be attributed to any lesion of the nervous system produced at the operation. Death is dependent solely on the removal of the gland: because, first, if a fragment of the gland of a certain size be left, death does not ensue; secondly, if a portion of the gland be successfully grafted in the subcutaneous tissue, and if the graft acquire proper vascular connexions, the entire normal gland may be removed without fatal consequences; thirdly, in some cases of the removal of the gland survival is prolonged by the administration of an extract prepared from the gland substance.

Thyroid.—The complete removal of all thyroid tissue is fatal in all animals hitherto experimented on, and in man. The removal of the thyroid body, however, is not necessarily fatal, since thyroidal tissue may be present in certain bodies known as accessory thyroids and parathyroids. Accessory thyroids have the same structure as the thyroid itself, but the minute anatomy of parathyroid tissue is somewhat different in that the cells which enter into this tissue are not arranged to form the lining membrane of a colloid-containing vesicle; in the parathyroids the cells are arranged in columns resembling those of a compound tubular gland, there are no large alveoli and but little colloid. The parathyroids are sometimes—as in the rabbit—at some little distance from the thyroid itself, sometimes—as in the dog—embedded on the

surface of each lobe of the thyroid, and sometimes—as in the monkey—embedded in the substance of the thyroid itself.

Parathyroid and thyroid tissues do not play an equivalent part in preventing the development of the symptoms which follow thyroidectomy. In the rabbit the thyroid itself can be removed completely without permanent ill effects, as the parathyroids are left untouched by any such operation. Whether in the dog complete removal of one lobe together with the greater part of the opposite lobe leads to survival or death depends upon whether one parathyroid is left or not; that is, if one lobe be entirely removed life may go on in the dog after excision of the bulk of the opposite lobe, provided the upper extremity containing the parathyroid be left: if, however, this upper extremity be removed a much larger fragment of the thyroid (amounting from a half to two-thirds of the lobe) must be left. On the other hand, the complete removal of the parathyroids only is followed by no obvious ill effects in many instances, but some observers have described tetany and tremors. Thyroid grafts prolong life after complete thyroidectomy; but it is remarkable that the injection of thyroid extract does not, in the monkey at any rate, prevent with certainty the development of the specific symptoms seen after this operation. In dogs, also, thyroid-feeding after thyroidectomy only secures a brief prolongation of life; in but a few cases does it prevent an early death. Complete removal of all thyroid tissue is followed in the acuter cases by the rapid appearance of a series of symptoms of which tremor, tetany, clonic spasms, and dyspnoea are the chief; in other cases, in which survival is more prolonged, a curious swelling of the parotid and other regions ensues, the hair becomes brittle, and more or less complete alopecia results. Clonic spasms are most marked in the carnivora, and in them the operation is followed by death, usually within a few days. The clonic spasms are accompanied by dyspnoea with paroxysmal exacerbations. In the dog, albuminuria in considerable amount occurs also after the thyroidectomy. The group of symptoms produced suggests in many ways a toxaemia, and some authors have held that the blood is thus tainted; others, however, have failed to reproduce the symptom-group by the injection into normal animals of blood obtained from animals after thyroidectomy. In man removal of the thyroid is followed by a condition closely allied to myxoedema, if not identical with it; but not infrequently tetany also has been so produced. The experimental myxoedema, seen in animals surviving from the acute and initial symptoms, apparently depends upon a disordered metabolism, and this condition in the human subject can be arrested and held in check by the administration of thyroid extract; hence it is clear that whatever other functions the thyroid may possess it supplies at any rate a substance necessary for normal tissue-metabolism.

The thyroid tissue left after partial thyroidectomy undergoes a series of changes. The fragment increases considerably in size, but the structure of the normal thyroid is not accurately reproduced. The epithelium lining the alveoli proliferates, so that in places the alveoli are

no longer lined by a single layer of cells; on the other hand, there is no formation of colloid, so that the structure of the hypertrophied thyroid resembles the structure of the normal parathyroids.

The parathyroids after partial thyroidectomy undergo slight enlargement, but there is no abnormality in their structure and no formation of colloid.

The thyroid body contains a large quantity of a complex protein substance that contains, in addition to the usual constituents of proteins, a considerable proportion of iodine and some phosphorus. This protein body can be split up by various methods, amongst others by digestion, yielding on the one hand albumoses, and on the other a non-protein substance containing a considerable percentage of iodine—the so-called thyroiodin. It is probable that this complex protein substance, yielding this "thyroiodin" as one of its decomposition-products, is the main ingredient of the colloid matter present in the thyroid. Although the non-protein moiety of this complex substance contains a large quantity of iodine, a smaller proportion of iodine is also contained in the protein portion of the original colloid substance. The name "thyroiodin" was first applied to a non-protein substance, containing iodine, obtained by treating thyroids with strong acids; but this substance is probably identical with that described above as a decomposition-product of the colloid material. Both the protein moiety and the non-protein moiety of the colloid substance are described as "active," that is, they both produce the physiological and therapeutical properties of thyroid extract; but the non-protein substance containing the larger percentage of iodine is by far the more active of the two.

Formerly the colloid substance was looked upon more or less as an excretion; now, however, it is regarded as an internal secretion, and in confirmation of this the colloid has been found in the lymphatics of the gland, suggesting that it is being carried away to supply the requirements of the body, and perhaps more especially those of the nervous system. If the colloid substance containing this thyroiodin is the essential and specific substance elaborated by the thyroid, it is remarkable that life can be maintained, in the carnivora at any rate, when the parathyroids, or even one parathyroid is left; for these bodies contain but little colloid.

The Suprarenals.—Removal of one suprarenal in animals, according to most observers, is not followed by any serious or permanent derangement of health; but Tizzoni found that death followed unilateral extirpation not infrequently, and that the operation was followed by a series of changes in the central and peripheral nervous system [*vide* art. "Addison's Disease," Vol. IV. Part I.]. There is far less agreement as to the results of bilateral extirpation; some observers have found that death ensues within a few hours, others after a period of varying duration of cachexia and marasmus; others again state that complete bilateral extirpation is possible without either death or illness. It is possible that these differences, extreme as they are, may be due partly to the operative procedures adopted, and partly to the presence of accessory suprarenals. Those

authors who have alleged that bilateral removal of these glands is followed by death from marasmus have also described hypertrophy of the remaining gland after unilateral extirpation; and further, that successful grafting of an adrenal will prevent the fatal effects that usually follow bilateral extirpation; but it is said that, for the graft to be successful, it is essential that it should be derived from an animal of the same species.

Death after bilateral extirpation of the suprarenals has usually been preceded by a period of marasmus, often extreme; but in a few cases some observers have noted the appearance of pigment in situations normally more or less free from pigment. As in the case of the thyroid, so in the case of the suprarenals, it may be asked whether the fatal event be due to the damage done to the sympathetic nerves during the operation, or whether it be directly and solely dependent on the removal of the gland; and in the latter case whether death be due to the accumulation of some toxic material, or to the suspension of an internal secretion.

It is asserted that after complete removal of the suprarenals a condition of partial palsy supervenes, which depends upon the presence in the blood of a curara-like poison; and that if the blood of an animal in this condition be injected into a normal animal, it produces in the latter a similar train of symptoms. On the other hand, there is clear and definite evidence that the suprarenals contain, and probably secrete into the suprarenal vein, a substance capable of causing profound effects on muscular tissue, and more especially on the muscular tissue of the arteries. Suprarenal extract, when injected into the circulation, produces a sudden and great rise in arterial pressure, which rise depends upon contraction of the peripheral arterioles. This contraction of the muscular coat of the arterioles is brought about by a direct local action of this substance on the muscular coat, and not by any action on the vasomotor centre. This active substance, adrenalin, is a stable crystalline substance, contained most abundantly, if not exclusively, in the medullary portion of the gland, and its potency is such that an extract representing $\frac{1}{1,000,000}$ of the weight of the dried gland, when injected intravenously, will produce a very notable effect on the general blood-pressure. Adrenalin also produces glycosuria, when applied locally to the pancreas, and when injected repeatedly leads to extensive atheroma in the aorta. A substance closely resembling if not identical with adrenalin has been prepared synthetically, and like adrenalin produces constriction of the arteries and glycosuria. It is at least doubtful whether the phenomena alleged to follow extirpation of the suprarenals can be correlated with any failure of the circulation dependent upon the absence from the blood-stream of this pressor substance. Whatever doubt may remain as to the functions of the suprarenals, there can be no doubt as to the existence of an internal secretion; although perhaps it is not certain that the internal secretory activity of these glands is their sole function. There is some evidence that the cortex of the suprarenals is in some way connected with the growth of the body and the development of sexual maturity (5).

Pancreas.—The pancreas affords another instance of a gland in which

there is very definite evidence in favour of the existence of an internal secretion. In dogs the removal of the entire pancreas is followed by a condition of glycosuria, accompanied by great wasting and thirst, and ending soon in death—a condition far more entitled to the name of experimental diabetes than the more or less transitory glycosuria producible by that puncture of the bulb known as the "diabetic puncture." This fatal pancreatic glycosuria is not seen after ligature of the pancreatic duct, nor after removal of the bulk of the pancreas, provided only that a small fragment of the gland be left; but if this fragment be excised by a second operation, the fatal disorder ensues. For these reasons the glycosuria cannot be due to mutilation of the abdominal nervous structures, but must be regarded as due to the removal of the pancreas itself. The fatal effects following the removal of the pancreas can be prevented by the successful grafting of a portion of the pancreas in the subcutaneous tissue, just as successful thyroid grafting will prevent the fatal effects of complete thyroidectomy. Very exceptionally dogs have survived the operation of excision of the pancreas, and in such rare cases no glycosuria has ensued. The existence of experimental pancreatic glycosuria is unquestionable; the mechanism of its production is obscure. The hypothesis of the formation by the pancreas of a glycolytic ferment secreted not into the duct but into the lymphatics and blood-vessels, which therein assists in the decomposition of the sugar of the blood, although plausible, is not clearly established. (For islands of Langerhans, see p. 184.)

Pituitary Body.—The posterior lobe of this gland contains a substance that has a powerful diuretic action, and in addition has an action on the vascular system somewhat similar to that of adrenalin. It may therefore be said to possess an internal secretion.

Liver.—The sugar-forming function of the liver, that is, the conversion of the glycogen of the liver into sugar to be distributed to the body at large, is really an instance of an internal secretion. Some authorities, however, deny that the liver is a sugar-forming organ, and regard the glycogen as a precursor of fat; the great bulk of physiological opinion, however, is in favour of the former view.

Kidneys.—Experiments performed by myself have demonstrated that after removal of the greater part of both kidneys the excretory functions of the organ continue not only undiminished, but indeed that the amounts of urine and urea excreted may actually be increased in amount; and further, that this disordered metabolism may lead to great wasting and marasmus, followed by death. This disordered nutrition is entirely dependent upon the amount of kidney removed; hence it is possible that the phenomena are due to the arrest of a normal internal secretion. This result cannot be considered proved until the progress of the disordered nutrition can be arrested by the injection of kidney extract, or by the successful grafting of kidney substance. This, however, has not as yet been done successfully, and some observers have attributed the results merely to the disordered metabolism of starvation.

Sexual Glands.—The testes and ovaries produce, as is well known, a

profound effect in the economy; since not only the general nutrition, but also the whole or the main characteristics of the organism may be altered by the removal or degeneration of these glands. It is not known whether these glands produce their effects by the chemical stimulus of an internal secretion or by a reflex action on the nervous system. The removal of the organs, in the human subject at any rate, is occasionally followed by remote general effects which it seems difficult to attribute to the arrest of an internal secretion. Phenomena of this nature are the mental disturbance seen sometimes after the removal of the uterus, and perhaps the atrophy of the prostate that follows removal of the testes. It is possible that the removal of glands, like the removal of organs, may produce remote effects that cannot be attributed to the presence of an internal secretion; at the same time the remarkable discovery of the substance elaborated by the suprarenals shews what totally unexpected and profound effects an internal secretion may bring about (22).

The existence of an internal secretion in other glands is improbable. The removal of the salivary glands is not followed by any obvious effects. The spleen can be removed completely without ill results, although in certain cases the resistance of the animal to microbic infection is diminished. The complete removal of the pituitary body does not produce any obvious ill effects, at any rate within a short interval after the operation. It has been asserted, however, that the pituitary body undergoes enlargement after thyroidectomy, and thus some observers have considered that this body is accessory to the thyroid. The experiments of Schäfer and Oliver, however, indicate that the physiological action of the extracts of the thyroid and pituitary bodies may be antagonistic, not complementary.

Pathology of Internal Secretions.—The proof of the existence of internal secretions has given an impetus to pathology, and has led to the hypothesis that disease may be the result of one or more of the following variations:—(i.) The arrest, (ii.) the excessive formation, (iii.) the abnormal composition of an internal secretion.

In many cases these views of the pathology of diseased conditions are little more than surmises, in others they are more securely established; this is especially the case with reference to the diseases of the thyroid body. Such conditions as myxoedema and sporadic cretinism are held to be due to the more or less complete arrest of the internal secretion of the thyroid, and on the other hand exophthalmic goitre is thought by some pathologists to be due either to a hypersecretion or to an abnormal secretion of the thyroid. The view of the pathology of myxoedema is more securely established than that of exophthalmic goitre. Not only can a condition allied to myxoedema be produced by the removal of the thyroid in man and animals, but also the effects of myxoedema in the human subject can be removed by the administration of thyroid extract, and perhaps by thyroiodin. In the case of exophthalmic goitre the matter is not so simple; the phenomena produced by the injection of thyroid extract are not quite similar to those seen in exophthalmic goitre. That the

exophthalmos present in this disease may be unilateral has been urged as an objection to the view that it is produced by a poison circulating in the blood. However, metallic poisons, like arsenic and lead, will sometimes produce unilateral lesions of the nervous system, so that this objection is not a very powerful one. A more serious objection is that the general symptoms of the disease do not necessarily vary step by step either with the magnitude or with the rate of development of the goitre. Enlargement of the thyroid, very similar to that seen in Graves' disease, may exist without any of the symptoms of the latter malady. The changes in the thyroid body in Graves' disease are apparently very similar to those seen in animals after the removal of a portion of the thyroid. The enlargement is of such a character as to reproduce the structure of the normal parathyroid rather than that of the normal thyroid. The enlarged thyroid consists of alveoli lined with actively proliferating cells, and these alveoli contain no colloid. The fact that the thyroid gland in this disease has a microscopic structure analogous to that seen experimentally when a fragment of the gland is left and undergoes enlargement, has suggested to some observers that the enlargement of the thyroid in exophthalmic goitre is not primary, but secondary, and arises in response to some need of the economy. It is possible, however, that when the thyroid enlarges, whether primarily or secondarily, the form and structure of the enlargement might be similar. The clinical observation that exophthalmic goitre is sometimes followed by myxoedema certainly suggests that the former disease is dependent on the thyroid hypersecretion.

On turning to the other glands we find that the evidence available at the present time to prove that certain diseases are dependent upon disorder of the function of internal secretion is by no means so strong.

In Addison's disease the interesting observation has been made that the remains of the suprarenals found in this disease contain no active suprarenal extract. At present, however, it is not possible to give a complete explanation of the phenomena seen in Addison's disease on the hypothesis that all of these result from the arrest of the internal secretion. Yet it is possible that the weakness of the circulation may be due to the absence from the blood-stream of the powerful pressor substance elaborated by the normal suprarenal. Although some success has been achieved in treating cases of Addison's disease with suprarenal extract, no such striking results have as yet been obtained as those seen in the treatment of myxoedema with thyroid extract.

In the case of the pancreas and diabetes the present state of our knowledge is also unsatisfactory. It is certain that diseases of the pancreas may be accompanied by diabetes, but the association is not an invariable one. Certain pathologists have thought that lesions of the pancreas, especially destruction of the islands of Langerhans, are associated with a particular variety of diabetes running an acute course. Lesions of the pancreas cannot, however, be regarded as causing all varieties of diabetes, and it is probable rather that glycosuria is a symptom that can be produced by

various lesions in different organs. Treatment of diabetes with pancreatic extract or with secretin has been followed by very little success, if any.

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GENERAL PHYSIOLOGY AND PATHOLOGY OF DIGESTION

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THE process of digestion is in part mechanical and in part chemical. The first part of the process is required to reduce the less soluble portions of the food to a state in which the digestive secretions can act upon them. Moreover, it ensures the due propulsion of the alimentary mass, and the submission of it to the several agents which affect it in turn as it traverses the whole length of the digestive system. The chemical changes are effected by the action of various digestive secretions, poured out by special glands at different parts of the alimentary canal. These secretions subject the constituents of the food, chiefly the carbohydrates and proteins, to changes which convert them into bodies easy of absorption and fit for assimilation. So interdependent are the various parts of the process that failure either of the mechanical or of the chemical factors

leads sooner or later to disturbance of the whole. It is therefore convenient to consider separately the several portions of the digestive tract in which the chief alterations of the food-stuffs occur; yet, when use is made of such expressions as "oral," "gastric," or "intestinal digestion," it must be remembered that a disturbance of any one of these three processes will in more or less measure disturb the integrity of the rest.

I. Oral Digestion.—In the mouth the coarser portions of the food are broken up and subjected to a process of trituration, whilst the softer masses become more thoroughly disintegrated, and are incorporated with the saliva by means of the teeth, the tongue, and the buccal muscles.

The Saliva, as it appears in the mouth, is not the pure secretion derived from the three pairs of true salivary glands, but is largely mixed with mucus from the glands of the buccal mucous membrane, with portions of food in a state of decomposition, and with numerous micro-organisms. When, however, saliva is obtained pure, it appears as a frothy, viscid, and transparent fluid of a specific gravity ranging from 1·002 to 1·006. Its normal reaction is alkaline, but sometimes owing to decomposition it is acid. Under the microscope certain so-called "salivary corpuscles" are seen. These chiefly consist of altered leucocytes and squamous epithelium from the oral mucous membrane. Large numbers of micro-organisms are also visible. The average composition of saliva may be stated as yielding in 1000 parts: water, 995·1; solids, 4·9. The solids consist of about 2·9 of soluble organic matter and epithelium and 1·9 of salts. The soluble organic matter comprises a protein resembling serum-albumin, mucin, globulin, a diastatic enzyme (*ptyalin*), traces of a proteolytic ferment probably pepsin, and certain extractive matters, such as urea and leucin. Lactic acid is occasionally found, but is indicative of disease. The salts consist of the alkaline and earthy phosphates, carbonates and chlorides; sulphocyanate of potassium is an invariable ingredient of human saliva. Free ammonia can nearly always be found in oral saliva, being probably the result of decomposition. It is said that nitrates are also to be found. The three pairs of salivary glands are alone concerned in furnishing the diastatic enzyme of salivary digestion. A mixture of the secretions of all three pairs of glands is more active than the secretion of any one pair taken alone, but the secretions of these pairs differ one from another. Saliva from the parotid is rich in diastatic enzyme, but contains only a trace of mucin; the submaxillary secretion is rich in mucin, and contains but traces of the diastatic ferment. The sublingual saliva in man has not been investigated; but in dogs it has been obtained in a viscous mass resembling frogs' spawn. The physiological effects of the stimulation of the nerves supplying these two glands have been thoroughly investigated and are recorded in the physiological text-books. Here it is only necessary to summarise the results. Stimulation of the parotid by Jacobson's nerve produces a watery secretion containing little protein matter, diastatic ferment, or salts. Irritation of the sympathetic causes no secretion; but

irritation of Jacobson's nerve and the sympathetic at the same time gives rise to an abundant supply in which the organic constituents abound. The salts, however, are but slightly increased. Stimulation of the chorda tympani produces an increased flow of saliva from the submaxillary gland; if, however, the stimulation be directed exclusively to the sympathetic filament, the secretion, though abundant, becomes thicker and more gelatinous than that which flows on stimulation of both filaments. (For paralytic secretion, *vide* p. 258.)

(*a*) *Alteration of Reaction.*—The normal alkaline reaction of the saliva has an undoubted effect on the gastric secretion. Bernard found by experiment that it is the best stimulant to excite the flow of gastric juice, but according to the later researches of Pawlow the gastric secretion that follows the outpouring of saliva is due to the same psychical stimulus, namely, appetite. On the other hand, the anorexia that attends all cases of salivation may be due to the dilution and neutralisation of the gastric juice by large quantities of alkaline saliva. The reaction of the saliva is sometimes acid, even in persons apparently healthy. This is probably due to acid fermentation occurring in the mouth. It has repeatedly been stated that in diabetes the reaction is acid, and that this is due to the presence of lactic acid. Dr. Gamgee, from observation of two confirmed cases, in both of which he found a marked alkaline reaction, has denied this statement. Ralfe noticed an acid reaction on several occasions, but the phenomenon was not constant: when it occurs it is probably due to acid fermentation, and not to the secretion of acid saliva.

(*b*) *Alterations in quantity* are apparent in the dripping, moist, or dry, parched mouth, according as the secretion is profuse or scanty. The average daily quantity is about 1500 c.c. In normal circumstances an increased flow of saliva follows reflex stimulation by the sight and smell of food, but there may also be more direct causes. A form of salivation, which in its exaggeration resembles "the watering of the chaps" of a healthy appetite, is the profuse watery discharge which, with a similar discharge from the gastric glands, constitutes "water-brash." (For the causes of salivation, see p. 317.) The evils of long-continued salivation—weakness and emaciation—have been attributed to (α) a diminution of the amount of diastatic enzyme, which certainly has a low percentage ratio in this condition; (β) the drain of water from the body; (γ) the inhibition and neutralisation of the gastric juice by the constant swallowing of large quantities of alkaline saliva.

Diminution of the salivary secretion, as Cohnheim suggests, can only occur as a subordinate pathological incident. He points out that to stop all secretion either the destruction of all the glands or the destruction of all nervous influence upon them would be necessary. The only interpreting symptoms of which we have any positive knowledge is that pyrexia checks the parotid secretion. But even then the power of converting starch into sugar is never quite absent, and the diminution of the secretion is probably in respect only of its watery constituent. A

similar diminution of the secretion is seen in the thirst and dryness of mouth that attend any profuse drain of water from the system, as in diabetes. In this case the thirst is not caused by the presence of sugar in the circulation, but by the diuresis; for if we control the diuresis by opiates, the thirst and dryness of the mouth and the urinary excretion fall together, though the sugar excretion may be still excessive.

(*c*) *Activity of the Diastatic Enzyme, commonly called Ptyalin.*—In new-born children this ferment appears only in the parotid; it is not developed in the submaxillary gland till after two months of age. Recent investigations have shewn that the sugar which results from the action of saliva on starch is maltose; the reaction may be expressed by the following formula:—10 $C_{12}H_{20}O_{10}$ Starch + 8 H_2O = 8 $C_{12}H_{22}O_{11}$ Maltose + 2 $C_{12}H_{20}O_{10}$ Dextrin. Whether any sugar other than maltose is formed by the action of saliva on starch is not yet known. Dr. Sheridan Lea in his experiments did not obtain any positive evidence that such is the case. The diastatic action of saliva is proved by the disappearance of the starch and its conversion into sugar.

Alterations in Amount and Activity of Ptyalin.—As we are unable to obtain the ferment in an absolutely pure state, so as to determine it by weight, we have to ascertain its "diastatic value" by means of Sir William Roberts's method of "diastasimetry." This method depends upon the final disappearance of all dextrins (which are coloured yellow by iodine), the solution containing only maltose and achroo-dextrin, which are not so affected. This method of estimating the activity of a diastatic solution on a given standardised starch solution is probably the most accurate that we possess. The salivary ferment acts best in neutral solutions, but it is efficient also in alkaline and even in slightly acid media, when the degree of acidity does not exceed 0·023 per cent of mineral acid and 3 per cent of organic. Stronger solutions than these put a stop to its action. The ferment, therefore, retains its power of converting starch in the stomach so long as the gastric juice does not rise beyond a certain point of acidity. Even if the degree of acidity be so high as to arrest it, its activity may be restored by careful neutralisation. The action of the saliva on the starches varies with the amount of cellulose they contain, and also with their state of solubility; as, for instance, whether boiled or raw. Certain other substances hinder its action: tea, by virtue of the tannin it contains, has a strong inhibitory effect upon it; but the retarding influence of coffee and cocoa is but slight. Alcohol, if taken in large quantities, puts a stop to starch conversion. Sir William Roberts pointed out a rational way of dealing with the inhibitory effects of tea and alcohol by suggesting we should eat first and drink afterwards. There are certain drugs, also, which arrest and soon destroy the action of the salivary ferment: common salt, ammonium chloride, and some other substances, on the other hand, increase its activity. These points will be considered further on when the action of drugs on the salivary secretion is discussed more fully. The fermentative influence of saliva upon starch in the mouth is so very brief that we

shall defer a consideration of the results of the malassimilation of this substance until we treat of its further digestion in the stomach, and of the action of the pancreatic ferment in the intestine.

(*d*) *Mucin* is secreted in abundance by the submaxillary and sublingual glands; it gives to saliva its viscidity, and consequently aids in the act of deglutition. The secretion of mucus is increased when the mouth is inflamed, and in cases of mumps. Such catarrhal mucus contains an excess of albumin, lymph-corpuscles, and epithelial débris. The same variation is observed in the earlier stages of ptyalism. An increase of mucus, however, as Cohnheim points out, is not so important a factor in ill health as the opposite condition of diminution, since buccal digestion is not interfered with. A dry mouth, however, has always been considered a sign of serious illness. Fever patients, Cohnheim adds, can keep their mouths moist so long as they are freely supplied with water; a loss of the sense of thirst or inability to satisfy it is ominous of approaching coma. The mucin of the saliva has the further property of forming the peculiar stringy masses hawked up from the stomach in some forms of gastritis, chiefly the alcoholic; but this regurgitation may occur whenever acetic acid fermentation is set up by the ingestion of substances prone to give rise to it. From its occurrence in the early morning, after first rising, Frerichs was led to suppose that it is due to the action of free acetic acid upon the mucin of the saliva swallowed during the night, converting it into stringy and ropy masses. It speedily subsides on the administration of remedies which stop the fermentative changes in the stomach.

(*e*) *Salts.*—These chiefly consist of alkaline chlorides, mixed with earthy phosphates and occasionally also with carbonate of lime. Sulphocyanate of potassium is a variable constituent of the saliva. Samuel Fenwick made some important observations which point to this salt being a product of the secretion of the salivary glands, and found that it is much increased in quantity in gout and rheumatism, but deficient in chorea, acute atrophy of the liver, lead poisoning, and inanition. Other observers believe that the salt arises from decomposition in the oral and buccal cavities. The characteristic test for sulphocyanate of potassium is the production of a blood-red colour by the addition of perchloride of iron, which colour is immediately destroyed by a solution of perchloride of mercury.

(*f*) *Action of Drugs on Salivary Secretion.*—The salivary secretion can be increased, diminished, or altered in chemical composition by the administration of certain drugs: (*α*) An increase in the amount of saliva may be brought about, either in a reflex manner by means of such substances as ether, chloroform, acids, alkalis, mustard, pellitory, ginger, and horse-radish, which stimulate the mucous membrane of the mouth or stomach, or by the direct stimulation of the secretory nerves or of the glandular structures by such drugs as pilocarpine, nicotine, muscarine, physostigmine, mercury, and iodine; (*β*) in like manner the reflex excitability of the nervous centre controlling the secretion can be

diminished by opium, morphine, codeine, and bromides; or the nerve-endings in the glandular tissue may be paralysed by atropine; (γ) lastly, certain drugs are habitually excreted by the salivary glands; of these the most important are the iodides, which can be detected in the saliva within fifteen minutes of their entrance into the stomach. It is also noteworthy that both iron and quinine, when combined with iodine, are eliminated in the saliva. Pilocarpine renders the secretion more liquid, but at the same time diminishes its diastatic action.

(g) *Influence of Disease on the Salivary Secretion.*—In addition to the alterations in quantity and quality which the salivary secretion is prone to exhibit in various diseases, it is also found that the amount of the sulphocyanate of potassium contained in it varies considerably in different disorders. Thus, in the early stages of all acute febrile diseases, and in gouty, rheumatic, and lithaemic conditions, the addition of a few drops of perchloride of iron to the saliva produces an abnormally vivid reaction; while in cases of chorea, lead poisoning, severe jaundice, cancer and atrophy of the stomach, and marasmus, the salt may be entirely wanting, and fail to reappear until general improvement has set in (Fenwick).

II. Gastric Digestion.—**Process of Digestion in the Stomach.**—The food, more or less triturated and incorporated with the saliva according to the efficiency of the dental apparatus, passes through the pharynx into the oesophagus, and thence into the stomach. The time occupied by this passage is so short that hardly any conversion of starch into sugar can take place. Arrived in the stomach the alimentary mass is subjected to a kneading process, which still further subdivides its particles and, with the addition of the gastric juice and swallowed fluids, helps to reduce it to a liquid state. Some part of this matter is absorbed directly through the walls of the stomach itself, but the larger part, when thoroughly prepared, passes out through the pylorus into the duodenum as *chyme*. The duration of the digestive process in the stomach varies with the individual, with the kind of food, with the activity of the gastric juice, and with the contractile power of the muscular walls of the organ. Speaking generally, the process is completed in from five to seven hours; though from the earliest period of gastric digestion small quantities of the softer and more easily digested portions of the food pass through the pyloric orifice, leaving the more solid residue to undergo further kneading and rotation, and to come under the influence of fresh supplies of gastric juice. The chyme is an acid fluid which passes from the pylorus into the duodenum, there to be mingled with the alkaline secretions of the liver and pancreas. It consists of the various elements of the food, reduced to a soluble condition and altered by the gastric juice. These changes may be briefly summarised as follows: (a) The conversion of starch into sugar, begun in the mouth, is carried to a farther point; (b) Much of the protein and albuminoid material is converted into peptones; (c) Fat, by the digestion of its cell-walls, is partially emulsified; (d) Milk is curdled. The proper performance of gastric digestion may be disturbed mechanically

by a delay in the reduction of the alimentary mass to a fluid state, from feebleness of the muscular movements; by any obstruction to the onward passage of the digested products through the pylorus—such as stenosis of its canal, or thickening of the walls of the stomach itself; or by chemical changes, qualitative or quantitative, in the composition of the gastric juice, such as impair or hinder the process of solution. But although the factors of delay are chiefly concerned in the production of gastric derangements, yet those which unduly hurry on the food before its complete digestion also play some part in causing intestinal indigestion. These consist of an unduly active peristalsis of the muscular walls, or a feebleness, qualitative or quantitative, of the gastric juice. The *gastric juice*, which can only be obtained mixed with mucus, through a gastric fistula, has an acid reaction and a slightly acid taste; its specific gravity, when at first secreted under excitation, is 1·001 to 1·003, but becomes higher as the process of secretion continues, when it also contains more corpuscular elements. It has the property of converting protein bodies into diffusible peptones,—a property due to the combined action of pepsin and hydrochloric acid. In addition to pepsin, the gastric juice contains another ferment, namely, "rennin," a milk-curdling ferment. Besides these two ferments, and the acid which gives the secretion its normal reaction, the remaining constituents are of slight importance; they consist of mucus, fat, traces of organic acids resulting from digestion, and of certain salts, such as alkaline chlorides and earthy phosphates. From its natural antiseptic properties gastric juice exposed to the air retains its activity for some considerable time without putrefaction. The secretion is only poured from the peptic glands under stimulation; when digestion is complete no more juice is secreted. From this it would seem that the process of gastric digestion is immediately under the control of the nervous system. Pawlow has shewn by experiments in dogs that the desire for food, which is called appetite, is the most powerful excitant of the gastric secretion, and that the glands of the stomach commence to pour out their acid juice exactly five minutes after the first mouthful of food has been swallowed. Division of the two vagi completely and permanently abolishes the secretion of the stomach induced by this psychical influence. The same observer has proved the fallacy of the accepted teaching with regard to the effects of mechanical and chemical stimulation of the gastric mucous membrane, and has demonstrated that the principal local excitants of the gastric secretion are solutions of meat extract, water, milk, and gelatin. On the other hand, other forms of protein produce no secretion when introduced into the stomach through a fistula, while bicarbonate of sodium and fats actively inhibit the production of gastric juice.

Chemical Examination of the Contents of the Stomach.—Of late years much attention has been given to the variations in the quantity and quality of the gastric secretion during the course of different diseases; and the knowledge gained by the systematic examination of the processes of digestion in disorders of the stomach has not only resulted in the distinction of several forms of dyspepsia which were formerly included

under a single denomination, but has also brought to light certain facts of diagnostic and prognostic value. The following is a brief description of the principal methods which are in use at present for the chemical examination of the contents of the stomach.

The activity of the gastric secretion is at all times dependent upon the quantity and quality of the food. Thus, in order to obtain results which are capable of comparison, it is necessary in every case to administer to the empty stomach a meal the composition of which is constant and accurately determined, and to evacuate the stomach after a definite interval of time. For this purpose several "test-meals" have been recommended, of which the following are most commonly employed :—

1. A breakfast (8 o'clock) consisting of 70 grammes of white bread and 300 c.c. of weak tea. The stomach is emptied after an hour (Ewald).

2. A breakfast composed of 70 grammes of white bread and 500 c.c. of milk. The stomach is emptied after two hours (Klemperer).

3. A lunch (11 o'clock) composed of 150 grammes of bread, 80 grammes of minced meat, and half a tumblerful of cold water. The stomach is emptied after two hours (Germain Sée).

4. A dinner (2 P.M.) composed of 100 c.c. of soup, 60 grammes of beef-steak, and 50 grammes of white bread. The stomach is emptied after five hours (Riegel).

Of all these the most convenient is Ewald's breakfast, since digestion is at its height at the end of an hour, when the contents of the stomach are comparatively clear and easily evacuated.

Extraction.—The stomach is usually emptied by means of a soft india-rubber tube, which is passed into the organ and siphonage established by the act of coughing, combined with pressure on the epigastrium. In some cases, however, it is advantageous to extract the gastric contents by means of a syringe attached to the free end of the tube.

An hour after Ewald's meal about 30 c.c. of semi-digested material can usually be obtained. The mixture has a peculiar odour, like that exhaled by raw beef; but in cases in which fermentation has been active in the stomach the smell is sour or disagreeable.

Filtration.—The first procedure is to filter the mixture through paper. The rate at which this can be accomplished varies in different cases; it is rapid and complete when the material is diffluent and contains a large quantity of hydrochloric acid, but extremely slow if much mucus be present. The filtrate consists of a limpid, yellow, and slightly opalescent fluid possessing a characteristic odour and capable of resisting decomposition for some time. Under pathological conditions, however, the colour may be green from admixture with bile; or red or brown, if blood be present. In such cases also the odour may be sour (lactic acid), pungent (excess of hydrochloric acid, acetic acid), rancid (butyric), ethereal, saccharine, or offensive. The residue left upon the filter consists entirely of the materials which have escaped solution. If digestion has been active the amount is small, and the masses of bread appear

swollen and gelatinous; but if the gastric secretion has been deficient the residue is considerable in amount, and its several constituents are little altered in appearance.

Reaction.—The filtrate always gives an acid reaction to litmus paper. This may depend upon the presence of (*a*) hydrochloric acid, free or combined; (*b*) organic acids; (*c*) acid salts. The presence of a free acid can easily be determined by testing the fluid with a piece of congo-red paper, which, when brought into contact either with a mineral or an organic acid in an uncombined state, acquires a blue colour.

Total Acidity.—The next step is to ascertain the total acidity of the filtered fluid. This is readily accomplished by titrating 10 c.c. with the decinormal solution of caustic soda (4 grammes per litre) until neutralisation has been effected. The result can either be expressed in terms of hydrochloric acid, or more simply by the number of cubic centimetres of the soda solution which were required to neutralise 100 c.c. of the filtrate. Thus, if 7·5 c.c. of the alkaline solution were sufficient to neutralise the 10 c.c. of the filtrate, then the total acidity of the gastric fluid is said to be 75.

Under normal conditions the total acidity, after Ewald's meal, varies between 50 and 65. An excessive acidity usually indicates an abnormal amount of hydrochloric acid; a diminished acidity generally depends upon a deficiency in the secretion of this acid.

Qualitative Tests for the various Free Acids.—1. *Hydrochloric acid* in a free state is most easily recognised by Günzburg's test. A few drops of the filtrate are mixed in a porcelain dish with an equal quantity of a solution composed of phloroglucin, 2 parts; vanillin, 1 part; absolute alcohol, 30 parts. If free hydrochloric acid be present, on the application of heat a ring of a crimson colour appears around the site of evaporation, the intensity of the colour being directly proportionate to the degree of acidity. This test is an extremely delicate one, and is capable of detecting 0·002 per cent of (free) hydrochloric acid.

Boas recommends a test solution composed of resorcin, 5 grammes; white sugar, 3 grammes; absolute alcohol, 100 c.c. The test is applied in the same manner as the former one, but to produce the red colour evaporation must be continued to dryness. As the dish cools the colour disappears. This reaction is sufficiently delicate to reveal the presence of 0·005 per cent of the acid.

Many other colour reagents vary in tint in the presence of a free mineral acid; but they also react in a similar manner, though in a lesser degree, to organic acids. Thus congo-red, methyl violet, and benzopurpurin each become blue, while tropaeoline 00 turns a carmine-red.

2. *Lactic Acid.*—This acid is readily distinguished by means of the test proposed by Uffelmann. To 20 c.c. of distilled water containing one drop of the tincture of the perchloride of iron, 10 c.c. of a 4 per cent solution of carbolic acid are added; the mixture, now of an amethyst blue colour, on the addition of lactic acid will immediately change to a canary yellow. This reaction is a very delicate one, but is hindered by the

presence of phosphates, alcohol, glucose, or an excess of hydrochloric acid.

3. *Butyric Acid.*—This acid produces a turbid brown precipitate with Uffelmann's test, and possesses a characteristic rancid smell. If the filtrate be treated with an excess of ether, and the latter be separated and evaporated, the residue will contain the greater portion of the butyric acid which was originally present. If this residue be dissolved in water, and a few pieces of chloride of calcium added to it, butyric acid will separate in the form of oily droplets, which are readily recognised.

4. *Acetic Acid.*—To detect the presence of this acid a small quantity of the filtrate is extracted with ether, the ether is evaporated, and the residue is dissolved in water and carefully neutralised with the decinormal solution of soda. If any acetate of sodium be present the addition of a drop of the tincture of perchloride of iron to the solution strikes a blood-red colour.

Quantitative Estimation of the various Acids.—1. *Hydrochloric Acid.*—This acid exists in the stomach both in a free state and also combined with various forms of protein. It is necessary, therefore, to be able to estimate it in each condition.

The quantitative determination of free hydrochloric acid is most easily and quickly performed by means of a method introduced by Mintz. This method is based upon the rule that when a solution of soda is added to a mixture of mineral and organic acids the alkali will completely neutralise the former before entering into chemical combination with the latter. To put this method into practice, 10 c.c. of the filtered contents of the stomach are placed in a porcelain dish, and the decinormal solution of soda is allowed to fall into it, drop by drop, from a graduated burette. The mixture is repeatedly stirred with a glass rod, and from time to time a few drops are removed to a clean dish and tested with the phloroglucin vanillin solution. As soon as the latter fails to give the characteristic reaction it is obvious that all the free hydrochloric acid has been neutralised, and its percentage amount can readily be determined by multiplying the number of cubic centimetres of the soda solution which have been used by the number 0·365. It is wise to perform this estimation twice, and to take the mean of the two observations.

2. *Free and combined Hydrochloric Acid.*—Several methods have been devised for the determination of the total quantity of hydrochloric acid (free and combined) in the contents of the stomach (Hehner and Seemann, Sjqvist, Cahn and v. Mering, Leo, Hayem and Winther). The first method is the most convenient.

Method of Hehner and Seemann.—Ten c.c. of the gastric filtrate are exactly neutralised with the decinormal solution of soda, and the quantity of the alkaline solution used for this purpose is carefully noted. By this procedure all the acids present in the filtrate, both free and combined, are converted into the corresponding salts of sodium, so that the mixture contains only lactates, butyrates, acetates, and chlorides of sodium. The mixture is now evaporated to dryness and the residue collected,

placed in a platinum crucible, and carefully calcined. The incinerated material, which consists entirely of the carbonate and chloride of sodium, is dissolved in a sufficient quantity of distilled water, and to the solution is added an amount of the decinormal solution of hydrochloric acid exactly equal to that of the corresponding soda solution which was originally employed to neutralise the acid filtrate. A certain portion of this fresh acid is neutralised by the carbonate of sodium derived from the lactates and butyrates, while the surplus corresponds exactly to the amount of chloride of sodium present; that is, to the total amount of hydrochloric acid which was contained in the original 10 c.c. of the filtrate. In order to determine this quantitatively, the mixture is titrated with the standard solution of soda, and from the quantity of the latter required to effect neutralisation the amount of the mineral acid is calculated. Thus, if this second neutralisation has required 5 c.c. of the soda solution, the 10 c.c. of the filtrate must have contained $5 \times \cdot00365 = 0\cdot01825$ gramme of hydrochloric acid. In other words, the total quantity of hydrochloric acid present in the filtered contents of the stomach was 0·1825 per cent.

It has already been shewn that by the method of Mintz the amount of free acid can be measured separately; in order, then, to determine the quantity of hydrochloric acid which exists in chemical combination with the proteins of the food, it is only necessary to subtract the result of the first examination from that of the second. Thus, if in the case above-mentioned the free acid was found to be equal to 0·06 per cent, the amount of the combined acid must be 0·1225 per cent ($0\cdot1825 - 0\cdot06 = 0\cdot1225$).

3. *Lactic Acid.*—In order to estimate the quantity of this acid, 10 c.c. of the filtrate are boiled with a few drops of dilute sulphuric acid in order to coagulate any albumin which may be present. After filtration the fluid is evaporated until it acquires a syrupy consistence; it is then mixed with 10 c.c. of distilled water, and evaporated to dryness in order to expel all the volatile acids. The residue is extracted with an excess of ether, and the ethereal solution is separated and carefully evaporated. The solid material which remains in the flask consists entirely of lactic acid. This is dissolved in water and titrated with the decinormal soda solution until neutralisation is complete. Then, since 1 c.c. of the soda solution corresponds to 0·009 gramme of lactic acid, it is only necessary to multiply this decimal by the number of cubic centimetres of the alkaline solution which were used to determine the amount of lactic acid contained in the original 10 c.c. of the filtrate (Cahn and v. Mering).

4. *Butyric and Acetic Acids.*—The quantitative estimation of these acids has no practical value. However, it can be accomplished by the method described by Cahn and v. Mering, which has already been referred to.

Tests for the Gastric Ferments.—1. *Pepsin.*—The presence of the peptic ferment in the gastric contents is evidenced by the power of the filtrate to digest albumin. In order to determine this, a quantity of hydrochloric acid is added to 20 c.c. of the filtered contents of the

stomach, in order to endow it with a degree of acidity equal to 0·2 per cent. The mixture is then placed in a flask containing several small cubes of egg-albumin or shreds of fibrin, and set aside in a warm chamber. If the ferment is active the albumin will undergo solution, so that at the end of an hour its bulk will have appreciably diminished. The rapidity with which this change takes place affords a rough indication of the quantity of pepsin present.

A much more accurate method has been invented by Mett. Fluid egg-albumin is sucked into a fine glass tube of 1 to 2 mm. lumen, and coagulated therein at a definite temperature (95° C.). The tube is then cut into pieces, which are placed in a few c.c. of the fluid to be investigated and maintained at the temperature of 38° C. for a certain period. Solution of the protein occurs at the ends of the glass tubes. At the end of the experiment the length of the pieces of tube, and that of the undigested remains of the protein columns, are measured off with the aid of a micrometer scale. The difference gives the length of the digested protein columns in millimetres and fractions of a millimetre. By the researches of Schütz and Borrisow the relationship between the length of the digested column of egg-albumin and the pepsin-contents of the fluid under investigation can be expressed by the following law: the quantity of pepsin is proportional to the square of the rapidity of digestion, *i.e.* to the square of the column (expressed in millimetres) which the juice is capable of digesting in a given period of time. Thus, if one specimen of gastric juice digest a column of two millimetres of protein, and another a column of three millimetres, the relative quantity of pepsin in each is not expressed by the figures 2 and 3 respectively, but by the squares of these numbers,—*i.e.* by 4 and 9.

2. *Rennet.*—This ferment is always present in a gastric juice which contains pepsin. To demonstrate its presence 10 c.c. of the filtrate are carefully neutralised and mixed with an equal quantity of sterile milk. After exposure to a temperature of 38° C. for half an hour the milk will have undergone coagulation, while the mixture still retains its neutral or alkaline reaction.

The Soluble Products of Digestion.—In addition to the various acids and ferments which have been enumerated, the filtered contents of the stomach always contain some of the soluble products of digestion. Thus, small quantities of achroo-dextrin, maltose, and dextrose, resulting from the action of ptyalin upon the starch, can usually be recognised; while the action of the gastric juice upon the albuminous constituents of the food is indicated by the presence of syntonin, propeptone, and peptone. Minute quantities of fatty acids, arising from abnormal decomposition of the fats, may also be present.

The insoluble residue left upon the filter consists mainly of starch, cellulose, food débris of all kinds, fat, epithelium, mucus, and various forms of bacilli and micrococci.

Micro-organisms of the Stomach.—Numerous bacilli and micrococci are habitually present in the stomach of healthy persons; they gain

access to the organ either through the medium of the food or by regurgitation from the small intestine. Some of these are relatively inert, but the majority are certainly endowed with a specific fermentative action upon the various food-stuffs. Under normal conditions the growth and influence of these micro-organisms are controlled by the antiseptic properties of the hydrochloric acid of the gastric juice, which, in the proportion of 0·2 per cent, is capable of inhibiting most of the processes of fermentation. But in certain pathological states, either from a deficient secretion of the acid or an atonic condition of the walls of the stomach which permits stagnation of the food, bacterial decompositions are both numerous and active, and their products are responsible for most of the symptoms characteristic of dyspepsia.

Abelous isolated at least sixteen distinct species of micro-organisms from the gastric contents of healthy persons, including such diverse organisms as *Sarcinae*, *B. lactis*, *B. pyocyaneus*, *B. subtilis*, *B. lactis erythrogenes*, *Vibrio rugula*, *B. amylobacter*, *B. megatherium*. Several of these micro-organisms possess similar fermentative properties.

There are four principal forms of fermentation in the stomach :—1. *Lactic acid*, produced by the *B. lactis* and other varieties upon lactose and glucose. 2. *Butyric acid*, also produced by many different species of bacteria, but especially by the *B. butyricus*. This acid is formed from lactic acid, two molecules of the latter being transformed into one of the former, two volumes of H_2 and two of CO_2 being set free. 3. *Acetic acid*. The ordinary agent in the production of this acid is the *Mycoderma aceti*, but it may also arise as a secondary product of lactic fermentation. 4. *Alcohol*. This is occasionally manufactured in dilated stomachs by the fermentation of sugar. The *B. alcoholicus* is introduced into the organ in fermented foods and new bread.

Acetone is also sometimes encountered, while the action of the various bacteria upon the nitrogenous constituents of the food occasionally gives rise to forms of peptone with highly poisonous properties.

The Gases of the Stomach.—These are derived from (1) the atmospheric air ; (2) digestion and fermentation of the food ; (3) regurgitation from the intestines ; (4) escape from the blood-vessels of the viscus. (1) In healthy individuals the major portion of the gaseous contents of the stomach consists of atmospheric air that has been swallowed with the food, mixed with a small amount of carbonic acid derived from the carbonates of the saliva by the action of the acid gastric secretion. During the progress of digestion most of the oxygen becomes absorbed, the percentage volume of the carbonic acid is slightly augmented, while that of the nitrogen remains more or less constant until the chyme has been expelled into the duodenum. When effervescent drinks are indulged in, the gas eructated after the meal consists almost entirely of carbonic acid. (2) Diseases of the stomach, both functional and organic, are accompanied by gas-formation due to fermentation of the food. Many attempts have been made to analyse the gases eructated under these conditions, but the results obtained differ widely in the percentage

volumes of their various constituents. Thus, to judge by the tables published by Popoff, Frerichs, Ewald and Rupstein, Schülzen, and others, the percentage of carbonic acid varies from 13 to 26, of hydrogen from 21 to 32, of nitrogen from 33 to 47, and of oxygen from 6·5 to 12, with occasional lesser amounts of marsh gas and sulphuretted hydrogen. These figures are devoid of clinical importance, since the relative quantities of the different gases afford no help to diagnosis.

The investigations of Wilson, Pasteur, Jenner, Hoppe-Seyler, Friedreich, M'Naught, and Naunyn and Minkowski have proved that the several kinds of fermentation which occur in the stomach with the liberation of gas are due to the activity of micro-organisms that have gained entrance to it with the food, and are enabled to flourish owing to some defect of the secretory or motorial functions of the organ. It was formerly believed that the immunity from fermentation enjoyed by the healthy individual was due to antiseptic properties of the gastric juice, and in laboratory experiments it can be readily shewn that solutions of hydrochloric acid and artificial gastric juice are capable of inhibiting the development of yeast and other fungi. Kuhn and Strauss, however, have demonstrated that in some cases of gastric insufficiency even an excess of hydrochloric or lactic acid not only fails to restrain fermentation, but sometimes even promotes it. The gases that appear as by-products of the various fermentations have already been noticed, and it is only necessary to repeat that carbonic acid is evolved in considerable quantities during the lactic, butyric, and acetic processes, while hydrogen chiefly arises from the butyric fermentation. *Putrefaction* of the gastric contents is brought about chiefly through the agency of certain anaerobic bacteria, and its most important product is sulphuretted hydrogen. According to Lesage and Strauss this gas is produced by the *Bacillus coli communis* as well as by other micro-organisms. Boas demonstrated its existence in a number of cases of motor insufficiency in which free hydrochloric acid was present, and states that it frequently exists in the benign forms of pyloric stenosis, while lactic acid fermentation appears to be inimical to its development since it is never met with in cases of carcinoma. On the other hand, many authorities believe that the gas is derived either from articles of diet that have been swallowed in a state of incipient decomposition, by the reduction of sulphates or organic sulphur-holding substances, or by regurgitation from the bowel. Van Tieghem and Tappeiner state that the butyric acid bacillus is capable of forming both methane and sulphuretted hydrogen from cellulose.

(3) Incompetency of the pylorus frequently permits regurgitation of the intestinal contents into the stomach, where the alkaline carbonates of the bile and pancreatic juice are decomposed with the evolution of carbonic acid. Putrefaction of the albuminous constituents of the food constantly occurs in the small intestine, and hence in certain cases such gaseous products as marsh gas and ethylene find their way into the stomach, and when eructated are found to be inflammable (M'Naught, Hoppe-Seyler, Ewald, and Rupstein). In M'Naught's case, in which this

phenomenon was very marked, the composition of the eructated gas was atmospheric air, 9·2 per cent; CO_2, 56 per cent; H_2, 28 per cent; CH_4, 6·8 per cent.

(4) Very little is known concerning the escape of gases from the blood circulating in the walls of the stomach, but it is a well-known clinical observation that immense quantities of carbonic acid are often expelled from a stomach which is devoid of food. Thus, in certain cases of neurasthenia gastrica, gastroptosis, and also occasionally in hysterical individuals, a severe emotion or psychical shock will give rise to the eructation of immense quantities of carbonic acid, with visible distension of the stomach.

Clinical Study of Gas-formation.—The contents of the stomach obtained from a healthy individual exhibit no signs of gas-formation for forty-eight hours or even longer, but under pathological conditions bubbles make their appearance in the mixture within a short time, and a considerable quantity of gas may be evolved within twenty-four hours. This sign of fermentation is an important factor in diagnosis, and should be investigated in every case in which motor insufficiency of the stomach is supposed to exist. The most convenient procedure is the following:—A test-tube, completely filled with the gastric contents obtained by a tube or from vomit, is securely closed by an india-rubber cork through which passes a bent glass tube, and is inverted in a beaker. When the cork is inserted, some of the material is forced into the bent tube, and the test-tube is thereby rendered air-tight. The apparatus is very simple and easily cleaned, and permits the formation of gas to be readily observed. It occasionally happens that the stomach-contents are free from sugar, a condition which prevents the bacteria present in the material from causing the usual fermentation; it is then necessary either to employ a control tube to which a little dextrose has been added, or to add the sugar direct to the first tube. When these arrangements have been completed, the beaker is placed in an incubator, or in some warm place with a temperature of about 90° F. After each experiment all the parts of the apparatus must be boiled. If the test-tube be found to contain gas within a few hours, the case under observation is probably one of pyloric stenosis, since the higher degrees of motor insufficiency are always due to obstruction. If lactic acid exist in the gastric contents, carcinoma may be safely assumed to be the cause of the stenosis; but if free hydrochloric acid be present in excess, a simple ulcer, or its scar, has probably led to contraction of the pylorus. The non-development of gas until after the lapse of twenty-four hours usually indicates that the motor insufficiency is due to chronic inflammation or to some functional derangement of the stomach.

A qualitative analysis of the gases formed in the test-tube has no clinical interest, but the presence of carbonic acid is easily demonstrated by procuring its absorption by caustic potash, while sulphuretted hydrogen may be recognised by its smell and by its characteristic decomposition of lead acetate. Inflammable mixtures burn with a blue flame.

The Physiology of Digestion.—The introduction of food mixed with saliva is followed within five minutes by a secretion of gastric juice, which continues without intermission until gastric digestion is complete. But, although hydrochloric acid is secreted from the outset, the presence of free acid in the contents of the stomach can seldom be detected until after the lapse of a period of time which varies with the size and quality of the meal. Recent investigations have afforded a complete explanation of this peculiar circumstance. It has been shewn that a strong affinity exists between all forms of albumin and free acids, each variety of protein possessing a definite and constant acid equivalent. Thus, 100 grms. of milk are found to saturate or "fix" 0·298 grm. of hydrochloric acid, while the same quantity of meat requires 0·843 grm. of the acid; the resulting chemical compound is possessed of some stability, but, though acid in reaction, it never affords the colour reactions of a free acid. It may, therefore, be accepted that the contents of the stomach will appear to be devoid of free hydrochloric acid until the whole of the albuminous portions of the food has been completely saturated. It is important to notice that the strength, as well as the amount, of the gastric juice varies according to the form of protein introduced into the stomach. Thus, the greatest digestive power belongs to the juice poured out on bread, the mean proteolytic power of which is represented by 6·64 mm. A diet of flesh calls forth a juice of 3·99 mm. digestive power, and one of milk of 3·26 mm. On the other hand, the most active juice occurs with flesh in the first hour of digestion, with bread in the second and third hour, and with milk in the last hour. (Pawlow). These observations explain many of the symptoms in cases of myasthenia gastrica and subacidity when they are fed upon an ordinary mixed diet.

Variations in the Chemical Constitution of the Gastric Juice.—*Hydrochloric Acid.*—Sudden and transient failure of the secretion of this acid occurs in cases of overloading of the stomach with fermentable substances (*embarras gastrique*). Free acid is generally absent when the stomach, especially the cardiac portion, is the seat of carcinoma. It has recently been shewn that malignant disease of any part of the body is associated with very great diminution of the free acid (B. Moore) (*vide* p. 502). The total output of acid is found to be constantly diminished in cases in which the gastric mucous membrane is affected with catarrhal inflammation, cirrhosis, or fatty and lardaceous changes. It is also diminished in many instances of profound anaemia and neurasthenia. The disorder known as nervous achylia gastrica is characterised by a complete absence of the gastric secretion (acid, pepsin, and rennet).

An excessive secretion of hydrochloric acid (hyperchlorhydria), as the result of the introduction of food, is encountered in all cases of hyperacidity, whether dependent upon direct irritation of the mucous membrane of the stomach by the ingesta, or upon indirect irritation in disease of the central nervous system. Abnormal acidity is also present in most instances of hypersecretion and of ulcer.

The secretion of the *peptic ferment* is much more constant than that of the acid, and, even when the stomach is the seat of grave organic disease, the introduction of a dilute solution of hydrochloric acid will usually procure the elimination of pepsin. Indeed, it is only when the mucous membrane has been destroyed almost completely by chronic inflammation that this ferment is absent from the gastric secretion.

The *rennet* ferment is the most constant of all the active constituents of the gastric juice, and only ceases to be secreted when the stomach is the seat of chronic and severe atrophy, and in those rare cases of nervous achylia.

III. Intestinal Digestion.—Under normal conditions the digestive secretions of the stomach, and those which operate within the small intestine, are maintained in a state of physiological equilibrium, the entrance of the chyme into the duodenum provoking an influx of the pancreatic and biliary fluids to an extent exactly sufficient to neutralise the acid contents of the stomach, and to establish the degree of alkalinity in the mixture most suitable for the action of those ferments which complete the processes of digestion. A moderate increase in the acidity of the gastric contents is followed by an increased flow of the bile and pancreatic secretion; while in certain functional disorders of the liver (and perhaps also of the pancreas) hyperacidity of the gastric juice is occasionally encountered—a phenomenon which may be explained upon the hypothesis that a more active stimulus than usual is required to call forth the tardy secretion of the biliary fluid. It follows that the digestion and subsequent absorption of food in the small intestine depend not only upon the integrity of the liver and pancreas, but even to a greater extent upon the due performance of the functions of the stomach. Intestinal indigestion may, therefore, originate either as a primary or a secondary disorder; but sooner or later in every case an aberration of function in one portion of the digestive tract is followed by derangement of the other parts.

Bile.—The composition and amount of the bile is naturally difficult to estimate in man, but it can be ascertained in the case of a long-established biliary fistula. Even in this instance, however, the gall-bladder may store up a considerable quantity of bile before it is poured into the common duct. Physiologists have often wondered why carnivorous animals should possess such a receptacle for storage, and why most herbivorous animals are without it. But, clinically, it appears that it is not an essential organ, as the gall-bladder is often found obliterated or obstructed after death, although no bad symptoms had been observed previously. It would seem, therefore, that the bile which flows direct from the hepatic duct is the best to make observations upon.

Influence on Digestion.—Modern investigations have proved that the functions of the bile are intimately connected with those of the pancreas, and of much greater importance than was formerly supposed. One of its properties is to destroy the activity of the gastric juice in the

duodenum, not only by helping to neutralise its acidity, but by an actual destruction of the pepsin, which is particularly inimical to the pancreatic ferments. In addition to this, the bile produces a constant and decided accentuation of the activity of the pancreatic enzymes. This aspect is most pronounced in the case of the fat-splitting ferment, the action of which it increases two to threefold, less in that of the proteolytic and amylolytic, which are only increased about twofold (Pawlow).

Quantity.—According to Dr. Gamgee, who has made a critical analysis of the observations of different authors, those of Drs. Copeman and Winston and of Mr. Mayo Robson possess an importance which cannot be attached to any previously recorded. From these researches we may conclude that the amount of bile secreted by the healthy human subject, when reabsorption of the bile from the intestine is prevented, varies from 1 pint to 1½ pint a day; whilst the solids excreted in the twenty-four hours amount to from 3·5 drachms to half an ounce.

General Characters.—The bile as it flows from the hepatic duct differs from that retained in the gall-bladder. As it flows from the liver it is a thin transparent fluid of golden yellow colour, like yolk of egg, of bitter taste and alkaline reaction. In the gall-bladder and common duct it acquires a viscidity from the secretion of their mucous glands, and often loses much of its alkaline reaction. Such bile is destitute of odour, and has a mawkish, bitter taste due to the bile acids. The specific gravity of the bile varies considerably according to different observations. Frerichs gives it as high as 1·03, whilst the observations of Noel Paton and Balfour record variations between 1·054 and 1·008. This difference may depend on the fact that some of the earlier observers did not distinguish between the biliary fistula of the common duct and a fistula communicating directly with the gall-bladder; yet the specific gravity of the bile from the gall-bladder is always much higher than that obtained directly from the hepatic ducts. After being withdrawn from the body, bile has a brown-yellow tint, but by exposure to the air it gradually assumes a greenish colour, due to chemical alterations in the pigments.

Cholesterin ($C_{26}H_{44}O$) is a constant element of bile. According to Hoppe-Seyler 0·35, and to Frerichs 0·26 represents the amount in 100 parts, and later observers have not altered these figures. It is still doubtful whether to regard cholesterin, the chief fatty constituent of the bile, as formed in the mucosa of the gall-bladder (Naunyn) or as excreted from the blood, which was the older view and has recently received some support from Aschoff. In forming an opinion on this point we must bear in mind that in certain forms of liver disease, in which there is a great destruction of liver-cells, a variety of coma often occurs, which has been attributed to the retention of cholesterin in the blood (cholesteraemia). We have no evidence, however, that cholesterin has any toxic influence. Krusenstern injected from 0·005 to 0·045 gramme of cholesterin daily into the veins of dogs, and found the animals unaffected. Pagès arrived at the same results. Looking at the question from a

purely clinical side cholesteraemia would be expected in all cases of jaundice in which there is considerable reabsorption of bile. If the liver be the seat of the destruction of the blood-corpuscles, which contain nearly all the cholesterin found in the blood in the normal state, we might naturally suppose that the cholesterin of the bile, reabsorbed in a free state into the blood, would rapidly induce cholesteraemia; but as a matter of fact the phenomena attendant on this condition are not common in obstructive jaundice, however complete; unless there be also considerable destruction of liver tissue and disease of the kidneys. Again, in acute yellow atrophy, in which there is rapid destruction of the liver-cells but comparatively slight jaundice, the coma is preceded and attended by symptoms much resembling those witnessed when phosphorus is injected into the veins of animals, and in patients dying from acute diabetic coma.

From these considerations it appears possible that the coma attendant upon hepatic disease associated with considerable destruction of liver-cells is not due to the accumulation of cholesterin, but to a general increase of the excretory matters in the blood; and this supposition is strengthened by the observation that the condition never occurs unless there be also some impairment of the renal functions.

Cholesterin is insoluble in water and dilute saline solutions. It is soluble in ether, from which it may be obtained in an amorphous state after evaporation. Treated with hot alcohol it is deposited on evaporation in characteristic rhombic plates, with notched edges, which float on water. Cholesterin is soluble in solutions of the salts of the bile acids, of soaps and neutral fats, which are probably the factors that keep it in solution in the bile. The chief tests for cholesterin are—(i.) Ready solubility in ether; (ii.) When heated with nitric acid, the mixture gives off yellow fumes of cholesteric acid, and the residue, on the addition of ammonia, acquires a brick-red colour. The latter reaction is somewhat similar to that which occurs with uric acid, and has led to deposits of cholesterin in the cardiac valves and vessels of the brain being mistaken for uratic concretions.

Lecithin is an organic fatty body which exists in the bile in the proportion of 0·25 per cent. In combination with cerebrin it was supposed to form protagon, but Dr. Gamgee's researches seem to shew that the latter is a definite chemical compound. According to this author, protagon, although it cannot be separated by the action of solvents into non-phosphorised cerebrin and a phosphorised body, can nevertheless by the use of caustic baryta be made to yield non-phosphorised substances. The large percentage of cholesterin and lecithin in bile appears to denote that the liver plays an important part in elimination of the metabolic products of the nerve-centres. The other fatty constituents of the bile consist of saponifiable fats, especially oleates and stearates. The latter possess a certain amount of interest, since in combination with the earthy bases they form the crust of many of the biliary calculi. In the case of the large solitary stone, which sometimes completely fills

the gall-bladder, as much as two-thirds of the outer crust may be composed of stearate of lime.

Mucoid Nucleo-Albumin.—Bile owes its viscidity to this body, which it acquires during residence in the gall-bladder. Former investigations seemed to shew that the material to which the viscidity was due was mucin, but subsequent observation proved that the reactions did not correspond with that body. Following up the subject, Paijkull, under the direction of Hammarsten, discovered that it belongs to the nucleo-albumins, and that in character it is like the nucleo-albumin discovered by Hammarsten in synovial fluid. The methods for the separation of this body are distinct from those used for the precipitation of mucin, but as these are tedious, and of no use to the practical physician, the reader is referred to the excellent account of them given by Dr. Gamgee. That this mucoid substance is derived from the gall-bladder and ducts is proved by treating an aqueous extract of the secretion of these structures with acetic acid, when a body is obtained which in all respects resembles the nucleo-albumin obtained from the bile itself. Ordinary mucin exists, nevertheless, in small quantities in the bile, and is probably derived from the hepatic ducts.

Bile Acids.—In carnivorous animals taurocholic acid is often the sole bile acid, whilst in the herbivora glycocholic acid is met with almost exclusively. In man the glycocholic acid is the chief constituent, taurocholic acid being usually present in traces only.

Glycocholic Acid ($C_{26}H_{43}NO_6$).—Several methods of isolating this substance are described in the physiological text-books. Dr. Gamgee recommends Hüfner's method as yielding the best results, when successful; but, as he points out, an excess of taurocholic acid in the bile prevents the precipitation of the glycocholic acid. This, however, should constitute no practical objection to its use in separating glycocholic acid from human bile, in which taurocholic acid is present in small quantities only. Glycocholic acid separates out in fine transparent needles, which are highly insoluble in cold water (1-300), but less so in hot water (1-120). They are almost wholly insoluble in ether, but are freely soluble in alcohol and glycerin. They have an acid reaction and a bitter-sweet taste. Both the acid and its salts are dextrogyrous. Sodium glycocholate may be obtained by dissolving the acid in a solution of sodium carbonate, then evaporating to dryness, dissolving the residue in absolute alcohol, and adding anhydrous ether, when the salt separates out in fine transparent needles which are readily soluble in alcohol and in water. An aqueous solution is precipitated by neutral lead acetate, and its solution has a partial action on saponifiable fats. The test for glycocholic acid and its derivatives has long been known as "Pettenkofer's," the principle of which is the development of a fine purple colour on admixture of cane-sugar and the very gradual addition of strong sulphuric acid. Several modifications have since been introduced to prevent the carbonising of the sugar, which is very apt to occur if the sulphuric acid be added too hurriedly, or the temperature allowed to rise above 70° C. Thus, Dreschel has proposed the use of

phosphoric acid instead of sulphuric, and Mylius has suggested the adoption of furfurol in water instead of sugar; furfurol being a product of the action of sulphuric acid on sugar. A very useful modification of Pettenkofer's test was proposed by Mr. Francis, formerly demonstrator of chemistry at Charing Cross Hospital; but, as he did not publish it, it is but little known. A few pieces of glucose are taken and dried thoroughly (when anhydrous they are to be placed in a well-dried stoppered bottle, and kept in the drying-chamber till wanted); then a small powdered fragment is placed at the bottom of a test-tube, and strong sulphuric acid (1 c.c.) added very gradually; on the surface of this a small quantity of urine is poured, when the zone of contact of the two fluids will become opaque, and then bright red gradually deepening into purple. If large quantities are required for clinical purposes the glucose and sulphuric acid may be mixed together and kept in a stoppered bottle in a cool place. The mixture forms sulpho-saccharic acid, of a pale straw tint, which gradually blackens; but, if kept at a low temperature and in the dark, it may be preserved for three or four days. As many other substances—notably morphine, albumin, and the phenols—give almost similar reactions with Pettenkofer's test, it is useful to employ the spectroscope for the identification of glycocholic acid and its derivatives. *Taurocholic acid* ($C_{26}H_{45}NSO_7$) occurs only in small quantities in human bile, and is sometimes absent; but in the dog it is the only acid present: for experimental purposes, therefore, recourse must be had to the bile of this animal. For the preparation of taurocholic acid the bile must be freed from all traces of glycocholic acid by precipitation with neutral lead acetate, after which the taurocholic acid is precipitated by means of a solution of ammonia and basic lead acetate in the form of lead taurocholate. This precipitate is well washed, boiled with absolute alcohol, filtered, and then treated with H_2S. The sulphide of lead is separated by filtration, the filtrate concentrated and then mixed with an excess of ether. The final precipitate, which is syrupy at first, is gradually converted into a mass of needle-shaped crystals (Hoppe-Seyler). Tests for bile salts depending on the low surface-tension of urines containing bile salts have recently been introduced (M. Hay, O. Grünbaum). Taurocholic acid is less stable than the glycocholic; its salts are less unstable, but still are not so stable as the glycocholates. Although taurocholic acid is intensely acid, its alkaline salts are neutral, and are soluble in water and alcohol; their aqueous solutions foam like soap. Acidified with dilute hydrochloric acid, solutions of taurocholic acid and its salts precipitate albumin, acid-albumin, and parapeptone; whilst albumoses and peptones precipitate the acid. Both the bile acids yield by-products which, however, are not important clinically; the most interesting of them is *Cholalic acid*, which is not found in normal bile, but in bile that has undergone decomposition; it is said to occur in small quantities in the small and large intestines.

The bile pigments must be regarded as middle products in a series of reducing processes which convert the blood pigment into the colouring

matter of the urine. Fresh bile owes its golden-yellow colour to one of its constituents, bilirubin, which can be obtained in a pure state in the form of an orange-yellow powder endowed with a characteristic spectrum. By exposure to the air or to the influence of other oxidising agents, bilirubin can be converted through several intermediate stages (biliprasin, bilifuscin) into a green substance termed biliverdin, the spectrum of which is quite distinct from that of the original material. The presence of bile pigment is easily recognised by the display of colours which ensues upon the addition of fuming nitric acid (Gmelin). So far as our present knowledge goes, the pigments of the bile exert no influence whatever upon the processes of digestion.

Salts.—Besides the large quantity of alkaline sodium salts that are combined with the bile acids, bile contains a considerable quantity of sodium chloride and sodium phosphate, traces of calcium and magnesium phosphate, and also traces of copper. Iron in variable quantities is always found in bile ash, and this fact is rendered more important by the researches of Dr. William Hunter, who discovered small deposits of iron in the hepatic cells in diseases which lead to rapid destruction of the blood, such as pernicious anaemia.

The Pancreas.—The normal secretion of the pancreas is a clear, viscid fluid which possesses an alkaline reaction, and froths when shaken. It contains very few structural elements. Its most important constituents are alkali-albumin, small quantities of fats and soaps, a trace of leucin and tyrosin, and a comparatively large amount (1 per cent) of carbonate of sodium, and ferments. Its secretion is not a reflex act, as was formerly supposed, but depends on the acid of the gastric juice liberating "secretin" from the mucous membrane of the duodenum, which directly stimulates pancreatic secretion (Bayliss and Starling).

Action upon the various Food-stuffs.—On starch, whether raw or boiled, the pancreatic juice exerts a specific action, converting it into erythro-dextrin and achroo-dextrin, and finally into maltose and glucose. This diastatic property depends upon the presence in the secretion of a special ferment closely allied to ptyalin, and named amylopsin. From the juice, or by the glycerin method from the fresh gland itself, a proteolytic ferment named trypsin can be isolated; this in the presence of carbonate of sodium rapidly reduces albuminous substances to a soluble state. The first effect of the ferment is to convert the protein into alkali-albumin, the final stage of its chemical action being the production of peptone. Some of the intermediate compounds are capable of being changed by the continued action of the secretion into a series of by-products of which the most important are leucin, tyrosin, indol, and skatol. The pancreatic juice also coagulates milk by means of a special ferment, which can be chemically separated from trypsin. It differs from the latter in that it is able to exert its specific action in the presence of an acid. The action of the secretion of the pancreas upon oils and fats is to produce emulsification and finally saponification, and in this way to promote the absorption of these substances by the intestinal mucous membrane, and

its activity in this respect is very largely increased by admixture with bile.

Succus Entericus (Enterokinase).—The mystery which has so long surrounded the mode of secretion and action of the intestinal juice has to a great extent been cleared up by the researches of Pawlow and Schepowalnikow, Sawitsch, Litwarew, and Bayliss and Starling. The first-named found by experiment upon dogs that the addition of a small quantity of succus entericus to the juice obtained through a pancreatic fistula produced a great augmentation of the activity of all the ferments of that fluid, especially of the proteolytic, and that the application of the usual tests for ferment action shewed that the active principle of the intestinal secretion was itself a ferment. To this substance, which is capable of producing an immediate conversion of the various pancreatic zymogens into their respective ferments, its discoverers gave the name of Enterokinase. They have also been able to prove that the excitor of the succus entericus is the local action of the pancreatic juice upon the mucous membrane of the small intestine, and that, while the secretion of the whole of the small intestine promotes the activity of the fat-splitting and amylolytic ferments of the pancreas, that derived from the duodenum exerts a special influence upon trypsinogen. Delezenne and Dastre have endeavoured to disprove the fermentative qualities of enterokinase, but the later researches of Bayliss and Starling quite uphold Pawlow's original statements.

I. *Primary Intestinal Dyspepsia.*—This may arise from (*a*) changes in the quantity or quality of the bile; (*b*) an insufficient secretion of the pancreatic juice; (*c*) disease of the mucosa of the small intestine.

(*a*) Obstruction to the flow of bile into the duodenum is usually accompanied by certain symptoms which are included under the name dyspepsia. They comprise a sense of fulness or discomfort at the epigastrium after a meal, nausea, acidity, and flatulence. The bowels are confined, and the stools are hard and clay-coloured. These symptoms probably owe their origin to an excessive fermentation of the food consequent upon the loss of the antiseptic functions of the bile, and on the stagnation of the contents of the intestine from the lack of the same secretion.

Although it is true that persons with a biliary fistula may live for many years without suffering any deterioration of the general health, careful examination proves that the absorption of fat in these cases is considerably affected, as much as 55 to 78 per cent of the total quantity ingested being sometimes eliminated with the faeces. Very little is known concerning the alterations which take place in the quality of the biliary secretion. It is certain, however, that many substances which would act injuriously upon the organism are eliminated by the liver, and are thus discharged by the bowel. It has also been suggested that in some cases in which the bile contains toxic material the secretion may irritate the duodenum directly and lead to catarrh of the mucous membrane, and finally to ulceration.

(*b*) Carcinoma of the head of the pancreas, obstruction of its duct by a stone, or atrophy of its glandular tissue, are all associated with a diminu-

tion or absence of the pancreatic juice. Under these conditions it is sometimes possible to detect an excess of fat or of crystals of fatty acids in the faeces. According to the researches of Abelmann upon the digestion of food-stuffs after excision of the pancreas, starch is readily absorbed by the intestine, though only a portion of it is converted into sugar; about 44 per cent of the albumins is absorbed during the same period of time; while the greater portion of the ingested fat is split up into fatty acids which appear in the dejecta. Emulsions of fat in the form of soap and gum emulsions are not absorbed. These figures are all much more favourable when the pancreas is only extirpated in part, even though the flow of the secretion into the intestine be entirely prevented. It must be accepted, then, that the active agents of the pancreatic juice are capable on emergency of undergoing vicarious excretion with the other intestinal fluids.

(*c*) Diseases of the small intestine may be accompanied by dyspepsia if the lesion deprive the mucous membrane of its power of absorption. This is chiefly the case when the lining membrane of the bowel is affected with a chronic form of catarrh leading to atrophy, a condition particularly apt to occur in children placed under unfavourable dietetic conditions, and in chronic pulmonary tuberculosis (Fenwick).

II. *Secondary Intestinal Dyspepsia.*— Any form of gastric dyspepsia may give rise to derangement of the intestinal functions. (*a*) In hyperacidity of the gastric juice the abnormally acid state of the chyme may completely neutralise the alkaline secretions which it encounters in the duodenum and thus put a stop to the digestive functions of the pancreatic juice. The unaltered starch thus allowed to accumulate undergoes acid fermentation under the influence of the various micro-organisms which inhabit this portion of the bowel. The absorption of fat is also delayed. The loss of the laxative properties of the bile increases the existing tendency to constipation, and at a later period the irritation due to the abnormally acid contents of the intestine gives rise to chronic catarrh of the mucous membrane of the colon with dysenteric symptoms. (*b*) Deficiency of the gastric juice also exerts a detrimental effect upon the course of intestinal digestion, inasmuch as fermentation of the food takes place in the stomach, and the duodenum consequently receives a large quantity of an extremely acid and undigested form of chyme. The ultimate result is the same as in the previous case. (*c*) Atony of the stomach, with loss of power in its muscular walls, leads of necessity to stagnation of the food, acid fermentation, and, finally to the same effects upon duodenal digestion which occur in cases of hyper- or subacidity.

Faeces.—The average daily quantity of faeces passed by a healthy man is about five ounces, and constitutes nearly one-seventh of the total quantity of the food ingested. In early infancy this proportion is somewhat greater, but it declines considerably with the approach of old age. Diseases of the stomach associated with vomiting and loss of appetite are accompanied by a marked diminution in the daily output of faeces; but those disorders of the digestive system which are characterised by irrita-

tion of the intestinal tract are usually accompanied by the frequent passage of stools in which the liquid element predominates.

Consistence.—During the first six months of life the stools present a peculiar homogeneous, butter-like consistence; at a later period they assume the solid and cylindrical form of excrement which results from the use of a mixed diet. Increased fluidity of the stools rarely arises from insufficient absorption of the ingested fluids (lienteric diarrhoea); more often it is the result of an excessive secretion by the mucous membrane of the intestine due to catarrhal inflammation, ulceration, or purgative drugs. An abnormally dry or tenacious state of the dejecta, on the other hand, may result from the undue absorption of the fluid contents of the bowel during its protracted residence in the intestine (constipation), or from a lack of secretion on the part of the mucous membrane of the gut, which may be due to the astringency of such drugs as opium, bismuth, lead, and chalk. Sometimes the faecal masses present a uniform coating of thick mucus, or the excreta may consist almost entirely of this material. These abnormal appearances indicate a catarrh of the mucous membrane of the colon, and therefore frequently accompany functional disorders of the digestive organs, especially in early life. Finally, it may be observed that in those forms of dyspepsia in which excessive fermentation occurs among the contents of the gastro-intestinal tract, the stools may present a pultaceous, frothy, or yeast-like appearance.

The reaction of healthy stools is usually acid, but its degree depends to a great extent upon the nature of the food. Increased acidity accompanies an excessive secretion of the gastric juice (hypersecretion, hyperacidity), as well as those numerous forms of disordered digestion which are associated with fermentative changes in the contents of the intestine. Diminished acidity of the stools is sometimes observed in the diarrhoea of infants, and in that which accompanies cholera and enteric fever.

Colour.—The stools derive their yellow colour from certain pigments manufactured from the bile by a process of oxidation (stercobilin, hydrobilirubin). The intensity of the colour varies directly with the activity of the biliary secretion, and with the length of time the undigested portions of the food have been retained in the intestine. Pale or colourless faeces, on the other hand, indicate a deficient entry of bile into the bowel; and accordingly gray, white, or putty-like motions usually accompany those functional and organic diseases of the liver which are associated with a diminished activity of the hepatic cells, as well as those diseases which mechanically obstruct the bile-ducts. Green stools are frequently observed as a result of digestive disorders in infancy. The peculiar colour has been ascribed to the presence of a chromogenetic bacillus (Hayem, Lesage); but it appears to depend rather upon an alkaline decomposition of the contents of the small intestine (Pfeiffer).

Black coloration of the faeces occasionally arises when altered blood is present (melaena); but it is more frequently observed as the result of the administration of such drugs as bismuth, iron, and charcoal. Certain articles of diet and a few medicinal remedies are also apt to impart their

characteristic colour to the motions; thus, claret and logwood produce reddish or violet dejecta; coffee and beef-tea a brownish, and rhubarb and santonin a bright yellow tint.

Odour.—The faecal odour depends upon the presence of certain volatile products of food decomposition (skatol, indol). It is consequently exaggerated in all cases of intestinal indigestion associated with excessive putrefaction of the contents of the bowel, whether this depend upon a primary disorder of the stomach, liver, or pancreas. The sour smell of the motions in infancy is due to the presence of organic acids derived from fermentation of the milk sugar.

Chemistry.—Healthy faeces consist approximately of about 25 per cent of solids and 75 per cent of water; but the relative proportion of these two constituents varies considerably both in health and disease. Among the normal constituents must be reckoned such products of pancreatic digestion as leucin, tyrosin, indol, and skatol; butyric, propionic, lactic, and valerianic acids; alkaline phosphates and other salts; neutral fats and soluble soaps; urobilin and stercobilin, and small quantities of ferments the chemical action of which is similar to that of pepsin and ptyalin.

Microscopic examination of the stools seldom affords any trustworthy indication of the nature of the digestive derangement. It may be stated, however, that an excess of starch in the faeces indicates a defective action of the diastatic ferments secreted by the salivary glands and pancreas; while an undue amount of undigested proteids points to the functional failure of the gastric and pancreatic secretions. Blood, pus, mucus, foreign bodies, entozoa, and bacteria are easily recognisable in the stools.

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DISEASES OF THE MOUTH

By Walter G. Spencer, M.S.

A. GENERAL

I. Bacteriology and Pathology of the Mouth.—(*a*) Micro-organisms in the Mouth. (*b*) The Saliva. (*c*) Dentition. (*d*) Caries of the Teeth. (*e*) Mouth-breathing. (*f*) Oral Sepsis. (*g*) Prophylaxis—Hygiene of the Mouth.

II. Clinical Appearances or Semeiology.—Fur on the Tongue. The Tongue in Disease.

III. Eruptions.—(*a*) Erythemas: Wandering Rash or Erythema Migrans; (*b*) Lichen. (*c*) Black Tongue. (*d*) Discolorations. (*e*) Stains.

IV. Acute Stomatitis in Relation with Micro-organisms.—(*a*) Pyogenetic Cocci causing Septic Stomatitis. (*b*) Aphthous Stomatitis or Thrush. (*c*) Diphtheritic Stomatitis. (*d*) Acute Ulcerative or Gangrenous Stomatitis.

V. Chronic Stomatitis.—(*a*) Herpes of the Mouth—Exfoliative Stomatitis and Pemphigus. (*b*) Leukokeratosis or Leukoplakia Oris.

B. LOCAL

I. The Lips. II. The Gums. III. The Palate. IV. The Salivary Glands.—Ptyalism. Dry Mouth or Xerostomia. Inflammation, Acute and Chronic. Calculus Formation. Symmetrical Enlargements.

V. Tongue.—Congenital and Acquired Deformities. The Fraenum. Glossitis. Actinomycosis, Tuberculosis, Leprosy. Syphilis. The Base of the Tongue. Macroglossia. Nervous Affections.

A. GENERAL

I. Bacteriology and Pathology of the Mouth.—(*a*) *Micro-organisms found in the Mouth.*—In a healthy new-born infant the mouth is stated to be free from micro-organisms; very few are found in a breast-fed baby; in one fed by hand the *Bacterium lactis* may be found in the remains of milk. If with the first dentition the gums become swollen, and débris of food be allowed to collect, staphylococci, streptococci, pneumococci, and colon bacilli may be present. *Oidium albicans* may be present in the mouths of perfectly healthy babies, and in older children the short, slightly virulent, so-called pseudo-diphtheria bacillus. From the tartar and carious teeth of children may be isolated streptothrix, leptothrix, or comma-like organisms, spirilla, and fusiform bacilli. In the more or less neglected mouths of adults, in the fur on the tongue, in the tartar around and between the teeth and the gums, mixed with the débris of food and epithelial cells, there are many organisms, including those above

named. Under conditions of health, these micro-organisms live in the mouth as saprophytes, and exhibit little virulence when cultivated and injected into animals. The common pyogenetic organisms present are the *Staphylococcus albus* and the *Streptococcus brevis*, neither of which shews much virulence when inoculated into animals. The pneumococcus must be raised in virulence before it can affect animals. The colon bacillus and its allies are held to produce the gases which render the mouth foul, but usually the colon bacilli obtained from the mouth are innocuous. Before the *Oidium albicans* can attack the mucous membrane of the child there must be some slight injury or inflammation. Even the organisms which grow freely in foul mouths, *e.g.* fusiform bacilli and spirilla, are cultivated with difficulty, and do not set up gangrenous affections in animals. Hence it would appear that the activity of these micro-organisms is only one of the factors in the production of the various diseases of the mouth.

(*b*) *The Saliva.*—The saliva secreted by healthy salivary glands is sterile, in ordinary circumstances micro-organisms are only found near the orifices of the salivary ducts. The inhibitory influence exerted by the saliva on the growth of organisms in the mouth is chiefly mechanical; the organisms, being prevented from becoming fixed, are soon swallowed, and do not obtain the blood-serum necessary for continued multiplication. Any diminution in the quantity of saliva relaxes this restraint, and the growth of the micro-organisms is rapid. But so slight are the antiseptic properties of the saliva that an increased flow of saliva, whilst mechanically beneficial in washing away organisms and decomposing material, fails to check acute ulcerative processes. Further, tartar with organisms is prone to collect around the lower front teeth, although these are specially bathed in saliva. Phagocytic properties have been attributed to the younger cells which escape from the tonsil to form the "salivary corpuscles." The chemical constituents of the saliva, such as the sulphocyanate of potassium, can hardly exert any antiseptic influence. On the other hand, the only ferment in the saliva, ptyalin, has simply an amylolytic action, and does not produce acids.

(*c*) *Dentition* is a normal physiological process, and as such causes no pathological conditions. The erupting tooth is not driven through the resistant gum like a nail, but the gum slowly atrophies before the advancing tooth, without any inflammation. Whatever temporary relief may follow lancing a congested gum, a wound is made upon which organisms may grow, and through which absorption may take place, at a time when the child's resistance may be below the normal.

(*d*) *Caries of the Teeth.*—The production of acids by bacterial fermentation of the carbohydrates in the food is an important factor in the production of caries. The organisms may act partly by inducing an acid fermentation, which decalcifies the enamel, and partly by producing a proteolytic enzyme which digests the organic basis.

(*e*) *Mouth-breathing* is the starting-point of many ills. In mouth-breathers the mucous membrane becomes dry, and from the resulting

multiplication of micro-organisms the mouth becomes foul. As the result of disuse of the nose, the upper jaw, palate, and teeth are imperfectly formed; there are ridges on the palate, hypertrophy of the gums, and irregular crowding of the teeth, which soon decay. The muscles closing the lower jaw are kept relaxed, the depressors are in over-use, and a stupid expression results. The naso-pharyngeal hypertrophy causes deafness, and the deformity of the palate impairs articulation, hence the child falls behind in education.

(*f*) *Oral Sepsis.*—A healthy state of the mouth is of great importance. The local complications set up by carious teeth, and the pulmonary inflammations following operations on the mouth have received the attention of surgeons. Dr. W. Hunter has noted the connexion between oral sepsis and some forms of gastritis and of pernicious anaemia (see Vol. V.). Whenever the surface of the oral mucosa loses its epithelium, staphylococci and streptococci produce pus. The inflamed granulations around carious teeth or in the pouches between the gums and teeth, the so-called pyorrhoea alveolaris, and granulations beneath thick crusts of tartar, or underneath irritating tooth-plates, are constantly discharging pus. Complications such as periostitis, alveolar abscess, antral empyema, or follicles in the tonsil or pharynx add to the discharge. The continual swallowing of this pus with micrococci sets up gastritis, attended by mucous vomiting especially in the morning, and the constant septic absorption is followed by anaemia. Oral sepsis may also at any time be the starting-point of a spreading or general infection. Suppuration in the neck and septic pneumonia may result, and pleural, renal, endocardial, and bone infections have been attributed to oral sepsis.

(*g*) *Prophylaxis—Hygiene of the Mouth.*—In infants any mechanical injury which will impair the protection of the epithelial surface must be avoided, such as rough wiping or teats, which have given rise to the so-called Bednar's aphthae; and especially any interference with the fraenum based on the erroneous superstition about tongue-tie. After feeding, the mouth of the infant may be washed with teaspoonfuls of water, and any remains of milk gently wiped away by soft rag or a camel's-hair brush. The rag must be burnt after use, and the camel's-hair brush well washed, kept in 1-20 carbolic acid for the use of that child only. The careful feeding of the infant should prevent the formation in the mouth of lactic acid in sufficient quantities to irritate the gums, and should obviate gastro-intestinal disturbance, which by diminishing the secretion of the saliva removes the restraint upon the growth of organisms in the mouth. During the first dentition special care is required to anticipate any swelling of the gums, such as may arise from the impairment of the antiscorbutic properties of milk by overheating. As soon as the child is old enough it must be taught to breathe through the nose, and to clean the teeth with a suitable brush night and morning. In some cases tooth-powder or soap should be used on the brush; in others a strand of floss-silk should be occasionally passed between the teeth; if the gums swell around the teeth, the finger must press on the gum to

squeeze out the débris from the fold between the gum and the teeth. It is on the regular use of the tooth-brush and upon nasal breathing that the health of the mouth in the growing child depends. The teeth of all school children require systematic inspection, for sound teeth have been found in 15-20 per cent only, and, when necessary, dental treatment to prevent or arrest caries. A carious tooth is a focus of infection, and its cavity must be cleared and filled so as to last until the tooth is shed, or, if it cannot be properly stopped, the tooth must be removed. Such teeth have been preserved in the past for fear of disturbing the second dentition. But in reality much more damage is done to the permanent teeth by a focus of infection. Decay of the four permanent molars must be arrested by filling cavities and so preserving the bite whilst the jaw is still growing.

Much more attention should be paid to the mouth by adults; the regular use of the tooth-brush night and morning, the removal and cleaning of dentures at night, the filling of hollow teeth are often neglected, especially by smokers. The foul mouth of the adult mouth-breather or of the chronic alcoholic is a potential source of grave danger. If the mouth of a patient is not cared for, acute pneumonia may become gangrenous, or enteric fever may be complicated by suppurative parotitis, or an operation on the mouth may be followed by septic pneumonia. In order to clean a foul mouth add to powdered chalk chlorate of potash up to 50 per cent. It is not advisable to use chlorate of potash for children, nor to prescribe it to other than careful adults, as it may be swallowed in poisonous quantities. But nothing is better than chlorate of potash for loosening sticky mucus and concretions between and around teeth. For general irrigation of the mouth permanganate of potash forms the most powerful deodorant, and is free from any danger of poisoning. It acts favourably as an astringent to the gums. Boric acid lotion and the mouth washes in vogue, such as listerin, glycothymolin, and odol, have feeble antiseptic powers, but are pleasant to the taste. For dissolving sticky pus, and so clearing out pockets and sinuses, peroxide of hydrogen is most valuable. The undiluted solution may be syringed into the sinus or pouch, or it may be diluted and the mouth frequently rinsed with it. Sore and tender gums may be painted with tincture of iodine; painful excoriations are relieved by painting with 5 per cent nitrate of silver, by which a protecting film is formed until the epidermis is restored. The organisms in a hollow tooth may be destroyed by inserting a flake of cotton-wool squeezed out of pure carbolic acid, which at the same time will arrest pain. Any acutely ulcerating or gangrenous area must be painted over with perchloride of mercury 1 in 1000, or better, with the same strength of the rather less irritating bicyanide of mercury. Foul pockets beside the gums, after being wiped out with peroxide of hydrogen, or swabbed out with carbolic acid or bicyanide of mercury, are packed with iodoform gauze. Later, when an astringent is required, sulphate of iron may be substituted. These, combined with the active treatment of the teeth by scaling, filling, or

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extraction by a dental surgeon, are the general measures for arresting oral sepsis.

II. **Clinical Appearances or Semeiology.**—The oral mucous membrane, and especially that covering the tongue, exhibits changes which are symptomatic of disease elsewhere in a very limited and indirect manner only. The fulness of the tongue stands in relation to the arterial blood-pressure; it is firm in health, and becomes flabby and indented by the teeth when the blood-pressure falls. Its colour is due to the haemoglobin shining through the epithelium, and so it becomes pale in anaemia or discoloured by collections on its surface; the papillae adhering to them have a variable collection of fur. In infants there is hardly any fur, for the filiform papillae have not grown. Whenever the mouth has not been used for some time, as during the night, fur collects on the filiform papillae. Behind the circumvallate papillae where the filiform papillae are absent there is no fur. Immediately in front of the circumvallate papillae the tongue usually presents some fur, because this is the part of the tongue that is least pressed against the palate or scrubbed against the teeth. The fungiform papillae are smooth and fur does not adhere to them. They are usually sunk to the level of the tongue. When only a thin fur covers the filiform papillae, the fungiform papillae may shew through the fur, and produce the *stippled or dotted* tongue. This is common in children, in those taking liquid food, and is caused by slight disturbance with loss of appetite, or any factor diminishing the quantity of saliva. When the skin is injected, for example, in scarlet fever, the fungiform papillae share in the change and stand out above the level of the tongue as bright red points forming the *strawberry tongue*. The thick collection of fur in the *coated, furred, or plastered* tongue is due to excessive bacterial growth, for there is no evidence of an excessive epithelial desquamation, except in inflammation. The rapid multiplication of organisms forming a thick coat is chiefly due to the diminished saliva in febrile states. The tongue is moved less, there is loss of appetite, and liquids only are taken. Free eating and drinking overnight produce a thickly furred tongue next morning, as a result of slight gastro-enteritis which diminishes the salivary secretion; there is also a marked acid fermentation sufficient to cause a disagreeable taste in the mouth. A thickly plastered tongue covered with yellowish-white fur is seen in rheumatic fever. The distribution of the furring may not be uniform; the paralysed side of a tongue is furred from disuse, as is the side which, from neuralgia, a painful ulcer, or a sharp tooth, is not used in mastication. Oral sepsis, whether general or local, increases the thickness of the fur. A marked diminution of saliva, muscular weakness so that the patient breathes through the mouth which thus becomes dry, and a fall of blood-pressure produce a *brown, dry, and shrunken tongue*. If the tongue be first covered with a thick moist fur which is not detached, so that the mouth becomes foul, and diminution of the saliva and mouth-breathing supervene, dryness results and the thick fur becomes *brown and shaggy*, micro-organisms adhering to the inspissated mucus. If, however, there

is a rapid diminution of saliva and a weakened pulse from the first, the tongue rapidly dries and shrinks with only a thin brown fur covering it. The inspissated mucus with the fur forms black crusts or sordes, which also cover the lips and teeth, whilst the shrunken tongue becomes a hard immobile mass. This is seen in such various conditions as extreme thirst, acute diarrhoea, cholera, and septic peritonitis. In typhus fever the tongue is described as becoming *black*. If time allow, the dried surface cracks into longitudinal and transverse chaps, which may penetrate the epithelium. The later stages of enteric fever, heart and renal disease, pulmonary tuberculosis, delirium, and intestinal obstruction are thus marked. The *cleaning of the tongue* is the reverse of these processes.

In addition to the foregoing changes of a more or less rapid character, there are others of a chronic character. A *raw tongue* may be simply one in which the corneous layers of the filiform and fungiform papillae are unduly thin, without any other disturbance than perhaps increased sensitiveness, *e.g.* to the influence of tobacco smoke or hot food. On the other hand, the corneous layers may be unduly thick, giving rise to the *mammillated, tuberculated, crocodile, or toad-like tongue* (langue scrotale). Such a condition has been noted in several members of the same family. *The senile wasted* tongue is produced in old edentulous women and others by the shedding of the filiform papillae, leaving a few of the fungiform and circumvallate papillae outstanding on a smooth surface, a condition which may easily pass into a dry, brown, chapped tongue.

The *hectic tongue* is a bare, red, dry, shrunken, cracked tongue seen in long and exhausting illnesses, such as pulmonary tuberculosis, diabetes, empyema, chronic pyaemia, sprue, dysentery, and liver abscess. The causes noted above have been at work, and a slight superficial glossitis has supervened, detaching the epidermis and leaving an excoriated surface. In sprue the rawness of the tongue may quickly disappear, but a relapse is common. In *the dyspeptic tongue* a slight degree of inflammation has followed, partly from causes noted above, partly from reflex nerve irritation which will be considered under "Herpetic Stomatitis" (*vide* p. 311).

III. **Eruptions.**—(*a*) *Erythema, Wandering Rash, or Erythema Migrans.* —This eruption has received numerous adjectives, each suggested by the clinical appearance which the rash happened to have assumed, such as dotted, or spotted, circinate, annular, exfoliative. The more objectionable names are those which would connect it with skin diseases such as ringworm, pityriasis, lichen, intertrigo, eczema, desquamative syphilide. The ambiguous name geographical tongue likens it to the irregular outlines on a map. It is really a rare affection, generally seen in a weakly child suffering from some dyspepsia or recovering from an illness. The rash begins as a red spot on the dorsum of the tongue which becomes smooth by the filiform papillae shedding their epidermal tips, whilst the fungiform papillae are prominent and injected. The spot spreads to form a ring. In the centre the filiform papillae slowly regain their corneous layers, whilst further away the surface is bare, with the fungi-

form papillae standing out on a bright red ground; most external of all is a faintly yellow, or even a golden-yellow border. The ring spreads to the margin of the tongue, and it may extend to its under surface. Meanwhile other spots have been forming rings, which meet one another, and one may stop whilst the more vigorous spread into the area of the weaker ring. No sooner has the centre of the ring returned to the normal than a new spot may start again and spread to form a fresh ring. Thus, then, concentric and overlapping rings give the eruption its peculiarly variable character. The rash, however, gives rise to no symptoms, it is generally noted by chance, but when attention is paid to it some itching and salivation may be complained of. Its pathology is unknown; no parasites have been discovered; there is no evidence that it is syphilitic or even parasyphilitic. Indeed the chief point about the affection is that it should not be mistaken for a congenital syphilide. A leukokeratosis is very rare in children. In them the syphilitic lesion is generally in the form of a raised mucous patch with a greyish surface, which occurs along with a syphilitic rash on the skin. A wandering rash in an adult would be at once distinguishable by its constantly varying characters. The wandering rash persists on a child's tongue for weeks or months, and is quite unaltered by any application. All that is indicated, therefore, is to attend to the general health and keep the mouth clean.

Another erythema—Koplik's Spots—is dealt with in the article on "Measles," Vol. II. Pt. I. p. 388.

The erythema of the mouth and tongue of infants is a slight form of the conditions discussed under "Thrush" [*vide* p. 306].

Though the erythematous glossitis and stomatitis of adults may simply be the result of dyspepsia, Möller connected a particular form with the presence of intestinal worms in rheumatic patients. Millard noted submucous patches which were at once removed by salicylates. Some erythemas may be due to infection, others to nervous disturbance as considered under "Herpes," p. 311.

(*b*) *Lichen in the Mouth.*—This eruption is difficult to distinguish from syphilis, especially the late manifestations of congenital syphilis, and the eruptions belonging to the parasyphilitic class of Fournier. Mr. Hutchinson described four or five cases of elevated, abruptly margined patches covering the papillae of the tongue, and resembling drops of size, though no pellicle could be scraped off. In some cases there was no other disease and no obvious cause; in others skin eruptions, lichen planus, lichen ruber, urticaria, or intertrigo were present. In a man of twenty-nine with characteristic lichen planus on the trunk Hallopeau described absolutely indolent, dull white patches of thickened epidermis covering the papillae of the tongue; they resembled the skin eruption, and differed from leukoplakia in not being so glitteringly white, and in the absence of ulceration. In association with lichen ruber and planus of the skin Besnier described the occurrence on the tongue of slightly salient silver-white spots, some isolated, some in contact, flat or a little

depressed in the centre. Dubreuilh and Frèche describe an eruption covering the mouth and tongue, consisting of spots and rugosities which disappeared under arsenic. In a case under my care in which lichen planus has persisted on the skin and in the mouth, a patch on the tongue passed into leukokeratosis threatening epithelioma, and has been cut out.

(*c*) *Black Tongue.*—Synonyms: *The Hairy Tongue*, *Nigrities*, *Melanoglossia*, *Melanokeratosis*, *Hyperkeratosis linguae.*—A peculiar patch is produced by the corneous layers of the filiform papillae, which instead of being cast off are prolonged into hair-like processes. The patch is nearly always seen on the dorsum in front of the circumvallate papillae, where fur is almost always present. But the patch has been noted in front of or behind this area, or on some other part of the mucous membrane of the mouth. The patch may be sepia-coloured; it has even been called yellow or blue, but black is the common colour. In extreme cases the back of the mouth has been described as apparently full of waving hair, though the filaments are not true hair but much elongated filiform papillae. We are ignorant of the cause, and even as to the nature of the black pigment. The colour has been attributed to black spores, *e.g.* of *Aspergillus niger*, or to a peculiar discoloration of the epithelium. At present all the statements about parasites remain unconfirmed. As a rule the condition lasts a few days only, coming and going without any obvious reason. Other cases have persisted, and have been attended with pain or a mawkish taste. If scraped off the patch reappears; it is not influenced by any treatment, but ultimately disappears spontaneously.

(*d*) *Discolorations of the Mucous Membrane of the Mouth and Tongue* (1).—In Addison's disease more or less dark or black marks may appear on the mucous membrane of the lips, cheeks, tongue, or palate. Brown and bluish-black patches have also been noted in other exhausting diseases, such as those of the heart and kidney, emphysema and bronchitis, and in pulmonary tuberculosis without any implication of the suprarenals. Xanthelasma has been occasionally noted on the sides of the tongue, over the ranine vein, and on the palate. Blood-stains appear on the dorsum of the tongue and on the palate in infantile scurvy (103), and in purpura haemorrhagica.

(*e*) *Stains of the Mouth by Drugs, Dentifrices, Sweets, Food Materials, and Accidental Discolorations.*

Black.—Iron preparations, ink, dark red wine, mulberries, cherries, charcoal. *Brown.*—Coffee, tobacco, liquorice, prunes. *Brown-red.*—Chocolate, rosanilin, permanganate of potash. *Red.*—Acid nitrate of mercury, rhatany, cochineal, raspberries. *Yellow.*—Chromic acid, laudanum, saffron, rhubarb. *Blue.*—Indigo, aniline blue. *Yellowish-white.*—Nitric acid. *Brownish-white.*—Nitrate of silver. *Greyish-white.*—Sulphuric, oxalic, and carbolic acids, with shrivelling. *Dead-white.*—Corrosive sublimate. *White, with flakes of epithelium detached.*—Ammonia. Dyes from dentifrices, sweets, or fragments nibbled off from an aniline pencil may become lodged between the teeth and keep up the staining.

IV. **Acute Stomatitis in relation with various Micro-organisms.**—

The clinical varieties of stomatitis are many, and authors differ widely as to names, and may even contradict one another. Thus, the term Aphthous is used to denote the stomatitis connected with *Oidium albicans*, and also to denote stomatitis in which this organism is held to be absent. But a clearer distinction of the forms of stomatitis is becoming possible by noting the chief organisms found. Still it is impossible at present to be precise as to the share taken by the organisms in the causation of stomatitis.

(*a*) *Pyogenetic or Septic Stomatitis.*—Under the head of Oral Sepsis mention has been made of the various conditions which favour the growth of pyogenetic organisms. In its milder form *septic stomatitis* has been termed catarrhal, simple, or erythematous. It is attended by slight swelling, tenderness, and salivation. When rather more severe the epidermis is detached at certain spots as a result of the formation of vesicles or pustules, which rapidly lose their contents. These sodden spots of epidermis may fuse into patches, which being covered by remains of milk or other food have given rise to the term pultaceous stomatitis. In the more acute forms much fibrinous exudation is mixed with the detached epidermis so as to form a membrane resembling that produced in diphtheria, although this acute membranous stomatitis may shew staphylococci only. An acute ulcerative process may go on to form an acute abscess under the tongue, or a slower process produces a cold abscess. Staphylococcic infection may produce pustules in the mouth; herpes of the lip, as in pneumonia, has the same origin. The *Streptococcus brevis* is found in most mouths, and streptococci cause the acute swelling and oedema which follow a wasp sting. Erysipelas starting on the face may spread into the mouth. In the course of scarlet fever there is a slight inflammation of the mouth, followed by desquamation; exceptionally, this may become a severe oedematous stomatitis with glossitis and tonsillitis. The septic mouths of children are discussed in the following section on thrush. The sore mouths of lying-in women result from puerperal infection and the consequent diminution of the salivary secretion. The sore mouths and stomatitis formerly noted in camps and on ships were cases of oral sepsis in scurvy. Gonorrhoeal stomatitis is a rare complication of gonorrhoeal conjunctivitis; the cases have all been mild, and have yielded to applications of nitrate of silver. Continental writers describe the direct infection of the mouth by the gonococcus.

(*b*) **Aphthous stomatitis** or **Thrush** in relation with *Oidium albicans* (*Blastomyces albicans*, *Saccharomyces albicans*). — Synonyms: *Aphthae; Muguet; Mughetto; Soor; Schwämmchen.* The nomenclature of the Royal College of Physicians connects thrush directly with *Oidium albicans*; Mikulicz and Kümmel, and also Prof. Osler, distinguish between Aphthous stomatitis and that in which *Oidium albicans* is found.

Thrush should be defined as a septic stomatitis attended by the overgrowth of staphylococci and of the *Oidium albicans* from organisms previously latent in the mouth. In typical cases the oidium has been

readily demonstrated; in many, however, whether from defects of method or from the scarcity of the organism, this has not been done. In the latter event, staphylococci have alone been demonstrated. The names simple, catarrhal, erythematous, follicular, vesicular, parasitic, and mycotic stomatitis are here deliberately avoided. The *Oidium albicans* has been the subject of much debate, a history of which is given by Plaut. The organism is held to be allied to, but not identical with, the *Oidium lactis* and *Oidium vini*, with which, however, it may be readily confounded. Both in lesions and in artificial cultivations are found yeast-like cells and threads, and under certain conditions spores are produced. In lesions the proportion of cells to threads varies, usually both are met with; in the minority there are cells only, and very rarely threads alone. Cultivations when simply deposited on the intact buccal mucous membrane of an infant have failed to grow; it is necessary to remove the surface layers or to irritate the epithelium of the mouths of animals, in order to inoculate the organism. Inoculation is much more readily accomplished if the thrush cultivation be mixed with one of staphylococci. Cultivations injected into animals in sufficient quantity produce merely a local abscess; the thrush organisms do not grow. The thrush organism has not been proved to live elsewhere than in the mouth and in the vagina, especially of pregnant women. In both cases the mucous membranes may appear quite healthy. It is not known how the organism exists outside the body. A similar disease attacks calves and foals, also birds.

In the mouth the growth of the organisms begins in the superficial layers of the epidermis, forming dead-white spots varying from a point up to 1 mm. or more in diameter. The corneous layer of the epidermis is raised by exudation, and can be wiped off from the deeper layers, leaving an exposed raw surface, which is painful, liable to bleed, and is readily infected. In the detached epidermis are found the cells and threads, or cells only. Sections of the mucous membrane shew threads penetrating between and through the deeper epithelial cells, which they tend to destroy, into the submucosa. There the threads penetrate the capillaries, which become blocked and the circulation arrested. With this is a concomitant growth of staphylococci. Around the chalk-white spot is an inflammatory zone. Along the channels made by the threads micrococci penetrate, the deeper layers of the epidermis necrose, and an ulcer forms, or the extensive thrombosis in the capillaries of the submucous tissue produces sloughs. In severe cases originally discrete areas of thrush become fused, and the detached epidermis infiltrated by fibrinous exudation and covered with fur makes up a false membrane. In extreme cases diphtheritic organisms perhaps take part in the formation of the membrane, and, in the acute ulceration and sloughing, the spirilla and fusiform bacilli to be noted under gangrenous stomatitis or noma. The cells of the thrush organism resemble those of yeast, being oval, measuring 5-6 μ by 4 μ. By budding colonies are formed, from which grow out threads of variable length and thickness. The threads

have a double contour, containing droplets, granules, and vacuoles, and may end in conidia, or swell out to a bulb. The conidia shew three or four cells in a row, which are being divided off. The bulbous enlargements form a capsule containing spores, chlamydospores.

Infection.—Thrush is usually an infection of children brought up by hand, although it may appear in premature, weakly children at the breast. In private practice it is generally mild; bad and fatal cases mostly occur in foundling hospitals and orphanages, it may be in epidemics. It rarely attacks older children and adults, and is then chiefly seen in the subjects of diabetes, enteric fever, and in old age. The disease is started by some gastro-intestinal disturbance which diminishes the secretion of the saliva, and gives rise to a scorbutic swelling of the gums. Lactic acid, whether taken in sour milk or formed from the remains of food in the mouth, also irritates the mucous membrane. Or some part of the oral mucous membrane may be injured.

The appearance of thrush, therefore, is preceded by gastro-enteritis and dry mouth, or by a sore mouth and gums. Then there is seen on the tongue near its tip or edge, or on the inner surface of the lips or cheek, pure white, slightly raised patches, surrounded by a dusky red zone, which is in marked contrast with the anaemic mucosa of the rest of the mouth. The patch can be detached, not by washing, but by wiping, when an excoriated bleeding surface is exposed. Fragments of curdled milk left after vomiting can be washed off, leaving the mucous membrane intact. In the detached epidermis are found thrush organisms and threads, and staphylococci, or micrococci only. It is an infective stomatitis, whether the threads of the oidium are aiding the cocci to penetrate, or whether the staphylococci alone are active.

The symptoms vary from restlessness caused by pain to refusal to take food, with rapid exhaustion following repeated vomiting and diarrhoea, which are more dangerous to the child than the local extension of the thrush. In the rarer severe cases, such as are seen in the epidemics in foundling hospitals and orphanages, the patches coalesce to form a continuous creamy layer over the inner surface of the lips, cheeks, the whole tongue except perhaps the middle of the dorsum, and the soft palate. Thence it may spread to the pharynx and oesophagus and obstruct swallowing. The mouth contains ropy saliva, or dries and sordes form. Beneath, excoriations may go on to ulceration, and this to gangrene—or there is acute oedematous inflammation, especially of the fauces. Even in the worst cases thrush does not usually spread lower than the oesophagus; the diarrhoea and the irritation round the anus and on the buttocks do not indicate the spread of organisms along the intestines. Exceptionally, however, the stomach and intestines have been attacked. The larynx and the middle ear are very exceptionally invaded. Metastases containing the organisms have been found in the brain, liver, kidneys, and lungs. Surface excoriations on the genitals and nipples have been inoculated with the oidium. In exhausted adults and old people the mouth is particularly dry.

Prophylaxis.—Thrush is prevented by the general care of the child to avoid gastro-enteric and buccal irritation, injury and infection of the mucous membrane; by care and cleanliness to prevent all decomposition of food, and all chance of direct inoculation; when epidemics arise in orphanages the general management is at fault.

Treatment must be first directed to the arrest of the gastro-enteritis and to the general nutrition of the child. Locally the chief point to avoid is any roughness which may further injure the mucous membrane and spread the disease. The mouth is wiped out with the softest rag, which is then burnt, or by a camel's-hair brush kept apart for the child only and well disinfected. With it the mouth may be frequently painted with 1 per cent of permanganate of potash. When the epidermis has become detached, the raw surface may be touched with 5 per cent nitrate of silver, so as to form a protecting film until the superficial layers of the epidermis re-form. If the surface of the excoriation be greyish and ulceration threaten, the spot should be painted with 1 in 1000 bicyanide or perchloride of mercury. Severer cases resemble diphtheria, angina, and noma as regards local treatment. (For epizootic stomatitis or foot and mouth disease, see Vol. II. Part I. p. 806.)

(*c*) *Stomatitis associated with the Diphtheria Bacillus.*—True diphtheritic stomatitis, *i.e.* in which the *Bacillus diphtheriae* has been demonstrated, is, when secondary to diphtheria of the fauces, quite rare, and primary diphtheria of the mouth is very rare. The few cases of true primary diphtherial stomatitis which have been established have been relatively mild, with some induration of the submaxillary glands; but the disease has subsided without any complication supervening. In neither of Dr. Trevelyan's two cases, nor in Wharton's case on the tongue of a boy, was the source of the infection traced.

Membranous stomatitis is more commonly due to pyogenetic cocci, staphylococci, and streptococci, and appears in severe cases of ulcerative stomatitis. They are dangerous cases, in which difficulty of swallowing and breathing may be set up by the swelling of the tongue, and may terminate by rapid heart failure. When gangrenous ulceration has followed on diphtheria of the fauces, improvement has resulted from the administration of diphtherial antitoxin.

(*d*) **Acute Ulcerative and Gangrenous Stomatitis; Cancrum oris or Noma, and its relation with Micro-organisms.**—*Etiology.*—In connexion with ulcerative and gangrenous stomatitis, there have been found, in addition to pyogenetic cocci, various organisms, comma-like and fusiform bacilli, spirilla or spirochaetae, and threads of streptothrix. In regard to these observations there are two debatable opinions: (1) that all the forms are variants of one organism; and (2) that the concurrence of these organisms are in causal connexion with the disease. Dr. Lingard succeeded in inoculating animals, monkeys and calves, from human cases of noma. The spontaneous gangrenous stomatitis of the calf could thus be imitated by inoculating the animal from cases of noma. Also gangrenous stomatitis was set up in calves by inoculating from the

lungs of horses attacked by gangrenous pneumonia. Spontaneous gangrenous stomatitis in pigs caused the death of inoculated rabbits in 9-10 days with large areas of necrosis in the heart. In the tongue and cheek of the cases of noma, in the lungs attacked by gangrenous pneumonia, and in the inoculated animals he found a bacillus termed *B. necrophorus*. In the lesions it was found especially in the necrosing zone, where the organisms were in process of penetrating intermuscular tissue, capillaries, and lymphatics. The bacilli formed long threads and knotted skeins. Miller found in sore mouths, and where tartar had collected between the teeth, motile comma-like organisms, *Spirillum sputigenum* and *Spirochaeta dentium*. In 1884 Babes described fusiform bacilli in connexion with an epidemic of gangrenous angina; Perthes found streptothrix organisms; Plaut spirilla and spirochaetae; Vincent the concurrence of fusiform bacilli and spirilla. Vincent has also found these organisms in hospital gangrene, which he considered, as did Taupin in 1839, to be the same disease as noma, and also in mercurial stomatitis. Fusiform bacilli and spirilla have, however, been found associated in dirty mouths and in carious teeth, without inflammation; these organisms may be absent in noma, micrococci only being found. Moreover, Vincent has failed to inoculate these organisms; it has not, therefore, been proved that these organisms cause noma, although undoubtedly they multiply freely in any foul ulcer of the mouth. Hence the name Vincent's angina should be avoided.

Morbid Anatomy.—The disease starts in a breach of the mucous surface, especially on the inner aspect of the cheek, and spreads rapidly both superficially and deeply, so that the cheek is quickly perforated. On the surface is a foul, greenish false membrane; beneath this is a zone of gangrenous tissue, and beyond a zone of dense inflammatory congestion with foci of gangrene and loculi containing foul pus and organisms. Externally there is a zone of dusky oedema with which the tissues are becoming indurated.

Symptoms.—Cancrum oris or noma is characteristically a disease of children exhausted by measles or scarlet fever. It is attended by a low fever, a dusky swelling of the cheek, and foul breath. Owing to an absence of complaint from the exhausted child the disease may be overlooked for a day or two and the swelling of the cheek put down to toothache. If noted early the disease may be arrested, but if it has already spread through the cheek, or to the tongue, or jaw, it is only rarely that, however vigorous the local treatment, the child can survive the septic infection. In adults the gangrenous process may follow a slight lesion in a foul mouth, especially when affected by syphilis, or alcoholism, or when suffering from mercurialism. As an epidemic it has occurred in connexion with scurvy in camps, gaols, and ships. Adults may die rapidly from oedema of the larynx, or from infection of the heart muscle.

Diagnosis.—It is important to distinguish it from anthrax, from cases of acute lymphatic leukaemia with swelling and ulceration of the gums,

from cases of diphtheria followed by gangrenous ulceration, and from ulceration following mercurial stomatitis.

Treatment.—Good results have followed the excision of a gangrenous patch in the cheek, for the wound has not become reinfected, and an early plastic operation can be done to obviate the deformity.

For slighter cases the actual cautery is used to destroy the necrosing zone, and should any recurrence ensue, the cautery must be reapplied. After the cautery, and in still milder cases, the surface should be painted with 1 in 1000 bicyanide of mercury and the mouth frequently washed out with strong lotion of permanganate of potash.

V. Chronic Stomatitis.—(*a*) *Herpes of the Mouth—Exfoliative stomatitis and Pemphigus.*—The name herpes is here confined to affections of the mouth which appear to be based on nerve disturbance, as distinguished from lesions provoked by micro-organisms. So defined the affections are classed with herpes zoster as occurring in some terminal branch, the lingual or other twigs of the 5th nerve, and the further tendency of the most frequent form to recur. The term herpetic has been also vaguely applied to what has been described above under Wandering rash, and Pyogenetic stomatitis and Thrush. For Herpes labialis, see p. 314.

As with herpes zoster, so in the mouth in a characteristic case there is stinging or acute neuralgia in the line of a nerve, followed by the rapid formation of a vesicle on an inflamed base. The effusion into the epidermis raising it is so quickly followed by the escape of fluid that the stage of the vesicle is scarcely ever seen. The spot of epidermis raised is about 1 mm. in diameter, and when it is detached the tips of the papillae are exposed on an easily bleeding surface; smarting and soreness follow, and there is disinclination to move the tongue until the epidermis re-forms. In the characteristic cases of middle-aged nervous women, about the climacteric, who are out of health, the vesicles quickly disappear, leaving the epidermis unaltered. In three weeks or so there is another attack, and attacks may continue to recur unless the general health can be improved. In some cases the condition appears to depend upon reflex irritation; thus, it accompanies dyspepsia, especially that set up by alcoholism. Irritation by tobacco, a sharp tooth or plate, hot drinks, or spices may apparently produce herpes through nerve irritation. Herpes is liable to be complicated by septic infection of the raw surface exposed by the detachment of the epithelium, especially when the mouth is foul, and the patient exhausted. Exceptionally an aggravated variety of herpes occurs forming a marginal *exfoliative glossitis.* Along the side of the tongue large flakes of epidermis become detached, leaving extensive excoriations, and there is at the same time considerable swelling of the tongue with pain, salivation, and difficulty in mastication. But the attack usually subsides spontaneously, although it is very prone to recur. Transitional forms are seen between the mildest cases, and the most severe bullous eruptions or *pemphigus.* Whilst the bullae may be limited to the mouth, the skin may be also affected, and vegetations appear in the axilla and groin. Pemphigus vegetans may prove fatal.

The treatment of herpes is mainly general, with the exclusion of local causes of irritation. The spots as they form may be painted with nitrate of silver (10 per cent), to form a film, which may allow of the patient taking food. Arsenic is not likely to be of much service; it should only be tried for a short time and in small doses. For the severe exfoliative and bullous cases opium is the most serviceable remedy (80). The relapsing cases in nervous women require a prolonged change of air.

(*b*) *Leukokeratosis or Leukoplakia, also termed Leukoma, Psoriasis, Ichthyosis, Tylosis.*—The name Leukokeratosis implies that whitish patches are formed by the thickening of the superficial layers of the epidermis as the result of a chronic inflammation or hyperplasia. The mucous membrane attacked is especially that of the tongue and inner aspect of the cheeks, but no part of the mouth is immune; the gums, floor of the mouth, the palate, the lips, may all shew the same changes.

Pathology.—The disease starts by a chronic inflammation immediately beneath the epithelium; small round cells collect, and in the most chronic form fibrous tissue forms. This is followed by diminished blood-supply to the epidermis, and flattening of the papillae. The result is a smooth patch of thinned epidermis covering cicatricial tissue. The patch is more sensitive than the rest of the epidermis, but if kept from irritation becomes stationary. A more active inflammation causes an increased growth of the corneous layers of the epidermis, so that the mucous membrane comes to resemble the skin, even that of the palm. Or the individual papillae become covered by thickened epidermal layers, producing a rough or even a papillomatous surface. Thus, a patch may have a horny glistening white appearance, or a rough patch may be discoloured by a collection of fur between the papillae. The surface of the patch may seem entirely quiescent, and yet *epitheliomatous* changes may be progressing beneath. Any induration, formation of nodules, or signs of ulceration shew that cancerous changes have begun. Histological examination of such a patch after excision shews two changes in the deep rows of epithelial cells antecedent to the commencement of epithelioma, viz. keratinous changes in the cells, and a tendency to a concentric arrangement, even so far as to form a well-defined nest-cell within the normal limits of the epithelium; in addition there is a steady increase of small round cells in the submucous tissue. Sometimes the interpapillary processes retain their relative order for a time and simply grow straight downwards. But sooner or later the irregular downgrowth of flask-shaped and anastomosing processes shews that the cancerous stage has begun.

Causation.—Leukokeratosis in its various forms is due partly to local, partly to general, causes. Tobacco, carious teeth, irritating tooth-plates are the common local causes, and are sufficient to produce the affection, but syphilis, alcohol, and exceptionally lichen, contribute to produce the patches. The influence of tobacco is peculiar to the individual and is not related to the quantity or quality of the tobacco smoked, nor to the

manner of smoking it. The patches generally form where the irritation of the tobacco smoke has been greatest, unless there have been some antecedent dental or syphilitic lesion. Chewing tobacco may also produce patches, especially on the inner side of the cheek. In this country the practice is mainly confined to sailors, but it is common in some parts of the East, *e.g.* in Ceylon, where the tobacco is mixed with betel. Acquired syphilis is the chief general cause of leukokeratosis, although this has followed the congenital form. So important is syphilis in this respect that leukokeratosis in a man who has not smoked, and in a woman, suggests unrecognised luetic infection, possibly of extra-genital origin. In a few cases dyspepsia may possibly be the sole cause of the leukokeratosis, the effect being increased by hot fluids and spices. Patches have also been noted in the course of lichen planus. Cutaneous psoriasis is said not to be accompanied by a similar affection of the mouth. Opaline patches and a rugosity of the mucosa of the cheek arise in glass-blowers, but disappear when this occupation is abandoned.

The conditions in which a diagnosis of leukokeratosis may be wrongly made are :—(i.) Hypertrophied fungiform, circumvallate, or the foliate papillae at the side of the tongue in front of the palato-glossal fold ; also hypertrophy of the lymph-follicles in front of the epiglottis. Such patients come early, give an exaggerated account of their symptoms, and are dyspeptic or neurotic. Close examination will discover a regular enlargement of normal papillae : (ii.) Congenital thickening of the epidermis giving a nodular surface, forming the mammillated or crocodile tongue, or lingua plicata, or hypertrophied patches on the palate (*vide* p. 303).

A well-marked leukokeratosis may become *epitheliomatous* at any time ; this has been noted in a patch apparently quiet for twenty years, and may set in years after the entire cessation of smoking. It is therefore essential that a patch of leukokeratosis should be cured or excised. The first step is to remove all sources of oral irritation by attending to the teeth, stopping smoking altogether, and restricting alcohol to the smallest amount. Only in the early stages is it of any use to administer antisyphilitic remedies by the mouth. Patches of leukoplakia, even when due to syphilis, are, when of long-standing, so resistant that Fournier regarded them as parasyphilitic. Inunction of mercury ointment with the finger, or the painting of the patch with lotio nigra are the best remedies for syphilitic cases. When not due to syphilis, bichromate of potash solution (10 per cent) may be painted on. If the patch be quite smooth, supple, and free from pain, the patient should be warned to avoid all causes of oral irritation, and to report himself at once should any change occur. An indurated, nodular, warty, or ulcerating patch must either begin to disappear under treatment within three weeks, or be excised. Such a persisting patch is to all intents and purposes an epithelioma.

Local Diseases

The Lips.—A congenital *hypertrophy of the lip* may be seen in idiots, or it may be due to a lymphangiomatous or venous hypertrophy, or *macrocheilia*, which may coexist with other deformities. Acquired hypertrophy is often due to mouth-breathing; the lower lip pouts, dries, becomes fissured, and persistence of the fissures sets up inflammatory enlargement. Crusts form which on separation leave a raw bleeding surface. Such lips have been termed strumous, but tuberculous infection is rare. Another form of hypertrophy is due to the mucous glands which become so enlarged as to be felt, like small shot, or miliary tubercles. The dilated glands are seen through the epithelium as yellow dots (Volkmann). Or the excessive secretion of these glands cover the lip with yellow crusts, beneath which the lips are inflamed and ulceration may go on beneath the crusts, followed by scarring. Unna said that this condition was first shewn to him by Baelz, hence the latter's name is sometimes given to the affection. It has been seen with seborrhœa of the face. As the outcome of mouth-breathing, the lips of exhausted patients are apt to become covered with sordes, beneath which ulceration may occur.

The treatment in all such cases is to apply boric acid and lanolin ointment, over which in the bad cases is laid a thin strip of gauze. For the yellow crusts Unna advised painting with tincture of iodine.

Herpes of the lip often attends pneumonia and some other acute diseases. It also occurs in children and adults owing to impaired health and dyspepsia. A patch of vesicles occur with smarting pain, and this may leave dry crusts, which when roughly detached expose a bleeding surface. The affection is probably due to micrococci. A patch of vesicles may be protected by painting on nitrate of silver, or collodion; later the boric acid ointment is applied.

Perlèche is the name given to infectious sores at the angles of the mouth in children. They must be distinguished from syphilitic lesions. Only pyogenetic cocci have been found. The treatment is that of the general health with the local application of a 5 per cent solution of chromic acid.

Syphilis of the Lip.—A chancre on the lips may be due to the causes mentioned under syphilis of the tongue (*vide* p. 324). The sore is generally surrounded rather widely by œdema. The occurrence of rapid œdema of a lip, not due to a sting or to acute erysipelas, should raise the question of a chancre. Eventually diagnosis is easy, for induration and swelling of the glands in the neck and a rash on the trunk invariably follow.

The other common syphilitic lesions are the mucous patches, cracks, and ulcerations about the angles of the lips which occur both in inherited and acquired syphilis. They are very infectious.

Leukokeratosis of the lip is especially due to tobacco smoked through a pipe with a hot stem. The result is a dry scab with a little fissure

beneath; in other cases the change spreads superficially along the prolabium. Sooner or later induration extends into the lip, a papillated or ulcerated swelling forms, and the submental glands enlarge, shewing that epithelioma has commenced.

Epithelioma in its most characteristic form occurs on the lower lip of an old man who has smoked, but it may be met with on the upper lip, and in a woman who has never smoked.

The Gums.—(*a*) *Hypertrophy of the gums from local causes* may be due to a carious tooth, and to organisms collecting between the tooth and the gum (see Oral Sepsis, p. 300). The irritation of a tooth may produce inflammatory hypertrophy of the alveolar periosteum, so that a fibrous epulis forms, or if it grow from the alveolus, a myeloid epulis. General hypertrophy of the gums may be due to widespread dental disease, especially when organisms and pus in pouches form between the teeth and the gum—the so-called pyorrhoea alveolaris. A more deep-seated general hypertrophy is congenital, although it may be first noticed at the first dentition, and it is then necessary to shave off not only the gum but the portion of the dental alveolus from which it grows.

Hypertrophy of the Gums from General Causes.—In scurvy the gums become swollen with bluish oedema. They easily bleed, and if neglected foul ulceration ensues. In babies affected with scurvy the gums are not usually attacked before teething. On the other hand, marked swelling of the gums has been noted before the teeth are quite through and with few signs of scurvy elsewhere, except haemorrhages into the palate (103). Swelling of the gums and of the palate are noticeable later, especially in backward children. Acute swelling and bleeding from the gums occur in leukaemia (see Vol. V.). The gums swell and tend to ulcerate from excess of mercury (see Vol. II. Part I. p. 1005). In lead poisoning the sulphide of lead is deposited in the superficial layers of the epidermis, forming a blue line (Vol. II. Part I. p. 1047).

Syphilitic sores and *leukoplakial patches* are relatively rare on the gums. *Tuberculous ulceration* of the gums is rarely acute, although it has been seen in the course of generalised tuberculosis. Lupus of the gums may arise by extension from the face.

Atrophy of the gums, sometimes called Rigg's disease, is a senile change. The gums shrink from around the neck of the teeth, which become loose and fall out. At the same time there is atrophy of the alveolar border, which in the upper jaw causes a disappearance of all the alveolar projection below the level of the palate. This change is very rapid in tabes in which a *perforating ulcer of the mouth* is attended by quiet necrosis of bone, so that a communication is soon formed with the antrum or nose.

The Palate.—*Abnormalities.*—The palate may be cleft in part or wholly. A bifid uvula, cleft of the soft palate scarcely invading the hard, and rare cases of holes with union in front and behind, are instances of partial cleft. Even if closed, the palate may be highly arched or shew a longitudinal ridge on each side; these changes are accompanied by mental defects, and special care is then required to see that the child

breathes through the nose, and articulates properly from the first. The epithelial pearls normally present at birth may be seen in excess along the line of the median suture. They are composed of epithelial cells, may cause a little irritation, but tend to disappear in two or three months.

Periostitis.—A boggy swelling, abscess and sinuses, as well as chronic thickening of the mucoperiosteum may be caused by the teeth, and limited or extensive necrosis, or empyema of the antrum, may follow.

Enlargements of the mucous glands of the hard and soft palate are set up by smoking.

Leukokeratosis results from the irritation of tooth-plates, as well as from syphilis and smoking (see p. 312).

Bednar's aphthae, or plaques ptérygoidiennes, are excoriations made by roughly wiping out the baby's mouth, especially over the hamular process of the pterygoid plate, or by a rough teat causing an abrasion on the hard palate. These sores become infected by staphylococci or the *Oidium albicans*. A rare case of invasion by *Aspergillus flavescens* is noted by Winfield. On the soft palate and uvula leptothrix may grow and produce white nodules or patches composed of corneous epidermis and the organisms, or even calculus may form, as in the tonsillar crypts.

The soft palate may present a fulness at the side because an accessory tonsil is contained between its layers, or because inflammation and abscess from the supratonsillar fossa have extended into it.

Syphilitic gummas, acquired or inherited, attack the hard and soft palate and quickly destroy it, in bad cases destroying the alveolar border of the jaw, or extending to the nose, and in limited cases producing perforation. When confined to the soft palate, adhesion to the posterior wall of the pharynx takes place and the pillars of the fauces are drawn inwards. As the result of perforation there is nasal intonation and food enters the nose. When there is closure of the nasopharynx the patient has a muffled voice, cannot blow his nose, suffers from Eustachian deafness and even from acute mastoid pain, owing to retention, and necessarily becomes a mouth-breather. An attempt may be made to arrest the process by anti-syphilitic remedies and local scraping, but usually fails completely. Open-air treatment is much more likely to succeed. Plastic operations may subsequently be done to close the perforation, or to release the soft palate.

Tuberculous destruction of the palate is rare as compared with the syphilitic. An acute ulcer with miliary tubercles round it may be seen on the soft palate, with signs of pulmonary tuberculosis. The chronic form of lupus has generally spread in from the face or nose, but it may begin on the palate, and may possibly be arrested by lactic acid, the cautery, *x*-rays, or radium treatment.

An extraordinary destruction of the palate and fauces was, after gradual exclusion of the ordinary causes, shewn by Mr. Tubby to be due to *glanders*, from which the patient finally died. Another anomalous case was proved at the autopsy to be *Mycosis fungoides*.

Benign tumours of the hard palate are composed of fibrous, glandular, and so-called cartilaginous elements, are encapsuled so that they shell out, and do not recur. They belong to the class of salivary-gland tumours described as endotheliomas. Fibro-angiomas and papillomas grow from the soft palate.

Melanotic sarcomas of the palate have often been recorded. Possibly the pigmented palate of Lascars and the patches seen in Addison's disease, also the pigmented palates of dogs, may have some indirect relationship. Starting at one point the growth rapidly spreads over the palate, the glands in the neck quickly enlarge, metastatic growths, which may not be pigmented, soon form. All the recorded cases have been quickly fatal, and it is useless for the surgeon to touch them.

Functional nervous disturbance causes twitching, choreiform movement, or spasms so as to produce a clucking noise. These disturbances may also indicate early stages of organic disease; it may be tabes, or allied degenerations. These lesions may be bilateral or unilateral. Unilateral lesions may be combined with paralysis of the vocal cord on that side, of the sterno-mastoid and even of half the tongue. Bilateral paralysis has been observed with pharyngeal paralysis, and also with an increased rate of the pulse from paresis of the cardio-inhibitory nuclei. The soft palate is frequently weakened or paralysed in diphtheria, but recovery follows. Enlargement of the tonsil and other swellings may by pressure on the nerves in the pharyngeal wall impair the movement of the palate.

The Salivary Glands. — *Ptyalism.* — Salivation means such an excessive secretion that the saliva escapes from the mouth, or is swallowed by a special effort. In bulbar paralysis, hemiplegia, melancholia, saliva escapes because it cannot be readily swallowed; this may be quite an early symptom of general paralysis of the insane. The formation of a quantity of viscid saliva is a special feature of hydrophobia; ptyalism is common in small-pox and pellagra. An excessive formation of saliva occurs in drinkers, the swallowing of which favours morning vomiting. A similar condition occurs in pregnancy, and may persist up to delivery; but it may also arise in neurotic women apart from pregnancy, and be attended with flushing of the face over the parotids. A flow of saliva is easily excited by ether vapour, by dentition, toothache, tic douloureux; it may also follow earache, due, it is supposed, to irritation of the chorda tympani. An extraordinary flow of saliva has followed a wasp sting in the mouth.

Mercurial Salivation (see Vol. II. Part I. p. 1005).

Slight cases of ptyalism may be relieved by injections of atropine, or by an alum or chloride of zinc mouth-wash.

Xerostomia is the name for dry mouth of nervous origin as described by Hadden. It occurs principally in neurotic women, past middle life and in poor health, and is the result of a diminished secretion and senile atrophy of the glands. In some there is also dryness of the conjunctiva from implication of the lacrymal glands. The affection occurs at a

later period than the atrophic rhinitis and pharyngitis connected with anaemia, in which the mouth and tongue do not share. Mental shock or worry, a vague illness, and carious teeth, have preceded the symptoms of dryness. The cases in which dryness has been followed by swelling and tenderness of the parotids will be considered below under Chronic Parotitis. Xerostomia may then be limited to cases of what is practically premature senile atrophy of the glands. The lips become dry and scaly, the tongue dry, smooth, distorted, the surface like parchment or crocodile skin, cracked, or fissured, the filiform papillae atrophied, the fungiform prominent. The gums, cheeks, and palate are dry, glazed, or encrusted with sordes. There is a salt taste in the mouth, but the taste for food is lost, and even a strong solution of quinine may not be recognised. The teeth are generally carious and slowly crumble away. Only a yellowish sticky fluid may appear in the conjunctival sac, and tears can hardly be excited; the nose may be dry and contain crusts, and the skin dry and harsh, but sometimes the patient sweats freely. The important point in the treatment is to remove all carious teeth and to fit in dentures which will prevent the mouth becoming dried by the air; in addition, steps must be taken to improve the general health.

Inflammation of the Salivary Glands.—It is now recognised that the infections of the salivary glands take place through the mouth by way of the ducts. The older views implied in the names "symptomatic," "sympathetic," "reflex," or "metastatic" inflammation may be dismissed as unproved. The term "secondary" parotitis should also be dropped, for although the inflammation follows changes in the mouth, the term has been employed in another sense. Virchow shewed in 1854 that the inflammation began in the parotid ducts and spread to the alveoli, and that only after their destruction does the inflammation, which may be purulent or not, spread to the connective tissue. Even in the case of gummatous and tuberculous disease of the parotid, it is the fibrous capsule in the one case, and the lymphoid tissue in the other, which is attacked, and not the parotid gland. Although the organism causing mumps is unknown, there can be no doubt that it attacks the glands by way of the ducts (see Vol. II. Part I. p. 586). Here attention may be drawn to anomalous forms such as symmetrical enlargements of the submaxillary, sublingual, and lacrymal glands, with or without enlargement of the parotid. Also with slight enlargement of the parotid, which may be overlooked, there may be acute orchitis with a temporary discharge from the urethra, acute ovaritis, or acute abdominal pain just above the umbilicus, attributed to pancreatitis, vulvo-vaginitis with oedema of the vulva, and mastitis. The swelling of the gland has given rise to facial paralysis which is partial and temporary, also to deafness, tinnitus, and vomiting from pressure backwards on the ear. A severe meningitis complicating mumps was only relieved by trephining and letting out some cerebrospinal fluid.

Acute and subacute septic parotitis is invariably preceded by changes in

the mouth. No organisms are present in the normal glands nor in the ducts, except at their orifices. The patient's lowered resistance and the diminished secretion of saliva allow of the growth of organisms which then creep up the ducts. In the presence of oral sepsis this occurs more readily because the organisms are the more virulent. The decomposition in the mouth is increased because the patient keeps his mouth open and is unable to masticate and swallow. Septic parotitis is a recognised complication of typhus, relapsing and yellow fever, dysentery, cholera, plague, especially during the second or third week of enteric fever, and less commonly in scarlet fever, measles, and small-pox. The dry mouth and the absence of mastication explain the occurrence of parotitis in the course of rectal feeding for gastric ulcer. In surgical practice it complicates all septic operations, but especially abdominal and pelvic genito-urinary operations, puerperal infection, and pyaemia. The onset is preceded by a fall of the blood-pressure, a diminution of the saliva, and also by the changes noted under Clinical Appearances (p. 302). With the onset of the parotitis there is increased fever, pain in the ear, limitation of the movements of the jaw, and a further diminution of the saliva. The swelling has usually the outline of the parotid, but implication of the deep process of the gland between the jaw and the mastoid process may at first escape notice. The swelling is more often unilateral and on the left side, or, if bilateral, the gland first attacked suffers most. The swelling may be doughy, diffuse, and the skin may become brawny. In severe cases the jaw can scarcely be moved, there are noises in the ear, and deafness. At first there may be much complaint of pain, but if the course be unfavourable, the patient passes into a drowsy typhoid state and dies. In favourable cases the swelling does not progress beyond painful induration, which soon begins to subside, and the mouth becomes moist. This grave complication is usually preventable, the care of the mouth being most important in all such acute affections (see p. 300). Hot fomentations are applied to the swelling, and incisions should be made into all brawny points, even before suppuration has set in.

Chronic parotitis is due to diminished secretion of saliva, which exposes the ducts to intermittent and partial obstruction by inspissated saliva. Experimental ligature of a salivary duct produces retention and cystic dilatation of the ducts, and swelling of the gland, which then atrophies as a result of pressure, and undergoes interstitial fibrosis. The dilatation of the secondary ducts may result in cysts, or the gland becomes an indurated mass of fibrous tissue. The intermittent character of the inflammation and its liability to recur may depend upon local factors, such as the valvular character of the obstruction, the shifting of a plug of inspissated mucus or of a calculus, or upon variations in the general disease causing the dry mouth.

Parotitis may be due to plumbism, and also to copper and iodide of potassium intoxication. In painters, attacks of chronic parotitis attend the other symptoms of lead poisoning; generally one parotid is especially indurated, and this may include Stenson's duct. In a marked case of

lead poisoning in a painter, described by Rénon and Labron, the submaxillary glands only were affected, Wharton's ducts escaping.

Glass- and trumpet-blowing may produce chronic swellings of the parotids and dilatation of the ducts. Air may enter the gland, causing emphysematous crackling; suppuration may follow. The cheeks become atonic and dilate in inflation like the representations of Tritons ("joues cassées").

Treatment.—General for the disease causing the dry mouth, and locally the elimination of all causes of oral sepsis. The enlarged gland, if painful, is fomented; if merely swollen, it is judiciously massaged to squeeze out the retained saliva.

Calculus formation in the salivary glands is due solely to local causes; there is no evidence of any general tendency such as is suggested by the word "sialolithiasis." A few cases follow injury, *e.g.* to Stenson's duct on the face, or are the result of cicatrices in the floor of the mouth. The impaction of a foreign body in Wharton's duct, although it has occurred, is not a common cause. The usual cause is spread of inflammation from a more or less foul mouth into the ducts. The organisms found in connexion with the calculi have been *Leptothrix buccalis*, streptococci, and, after suppuration, staphylococci. The submaxillary glands and ducts are by far the commonest affected; in Mr. Stewart's series of 89 cases, 73 belonged to the submaxillary gland and duct, 8 to the sublingual, and 8 to the parotid. As a rule a calculus, composed chiefly of calcium carbonate, is very slowly formed by apposition round a nucleus of mucus. It may grow to a large size before attracting attention, when the hard mass may give rise to the diagnosis of cancer. On the other hand quite a small concretion may be noted early because it so blocks the duct that as soon as the patient begins to masticate retention of the saliva to be secreted gives rise to acute pain and swelling. This swelling has then to be reduced by external pressure. In another group of cases chronic interstitial inflammation affects the whole submaxillary gland, and small calculi like grains of rice may be intermittently discharged. Chronic inflammation may necessitate excision of the gland; chronic interstitial inflammation will then be found with dilated small ducts and alveoli, the ducts containing plugs of inspissated mucus which form the nuclei for small calculi.

Treatment as for chronic parotitis, with the removal of the calculus, and if this fail, excision of the gland.

Symmetrical Enlargements of the Salivary and Lacrymal Glands.—This heading includes cases probably due to various causes. The condition is doubtless due to organisms, though none have yet been identified. Some of the cases have occurred in epidemics of mumps, and may be considered to be complications of that infection; others are cases of lymphadenoma or lymphosarcoma. In the special symmetrical enlargements described by Mikulicz, or *Mikulicz's Disease*, the swellings of the lacrymals and parotids were found to be due to small round cells, regarded as lymphosarcomatous and not as inflammatory. Mikulicz's

original case shewed the structure of lymphosarcoma. About two months later the man died from acute peritonitis after nine days' illness, during which the parotid swellings practically disappeared. Fuchs saw a similar case in which improvement followed the administration of arsenic, but the drug upset the patient, and fourteen months later the condition was unchanged. In Hattenhoff's case, an anaemic girl, aged 12, with phlyctenular conjunctivitis, enlargements of the lacrymals, parotids, submaxillaries, tonsils, and of the walls of the pharynx appeared. The swellings subsided under a good diet and the syrup of the iodide of iron. Adler's patient had enlargement of the lacrymal and salivary glands, and of the lymphatic glands in the neck. All these gradually subsided under arsenic, and four and a half years later he was well. Besides cases occurring in the course of mumps, others have apparently followed conjunctival irritation, by the dust of a flour-mill and of a cigar factory. Symmetrical swellings of the lacrymals, parotids, and sublinguals, as well as of the spleen and liver, have occurred in ordinary leukaemia (Gallasch). The symmetrical swellings have also been attributed to tuberculosis (Frost). In Snell's case of a woman aged 61, the swellings continued to increase slowly for five years, the swelling of the lacrymals reaching the size of a teacup. After this the tumour ulcerated, a perforation followed though the cheek into the mouth, and she died of exhaustion. This must have been an instance of malignant small round-celled sarcoma.

Cysts of the salivary glands and of the mouth, including *ranula* and *tumours of the salivary glands*, especially *endotheliomas*, are chiefly of surgical interest.

The Tongue. (See Clinical Appearances, p. 302.)—*Congenital deformities of the tongue* depend upon an arrest of development in connexion with the second branchial or hyoid arch. Hence an ill-developed lower jaw, or agnathia, is not necessarily associated with an abnormality of the tongue, although both the mandibular and hyoid arch may be ill-developed with a median cleft of the tip. The more marked deformities are seen in monsters, and if an abnormality occur in an idiot, or in a child with defective intellect and articulation, even although the tongue seem too fixed or too large, no good will result from a surgical operation.

The so-called *tongue-tie* is practically always an imaginary deformity, the tip of the tongue in front of the fraenum being somewhat poorly developed. Surgeons of large experience have never seen anything which may be called tongue-tie. Midwives should be punished for malpraxis if they touch the fraenum of babies, not only because haemorrhage and falling back of the tongue may result, but on account of the danger of septic infection.

Acquired deformities are now rare; they were commonly produced by the sloughing following the excessive administration of mercury, including that of calomel, to children.

Thumb-sucking is a vice in particular of idiot and mentally deficient

children, and along with finger and lip sucking may produce caries of teeth, deformity, and even dislocation of the jaw.

The tongue may be over-long, so as to be protruded like a dog's tongue; and *excessive mobility* may be acquired by curling back the tongue along the palate whilst depressing and elevating the jaw, until the tip can pass into the nasopharynx, so that the tip feels the orifices of the Eustachian tube and the posterior nares. Indeed, relief has been found in this way for intractable atrophic rhinitis and pharyngitis, for which it has been a traditional practice in India.

The Fraenum of the Tongue.—Ulceration of the fraenum may be produced during whooping-cough from the tongue being driven against the incisor teeth, and it may even be produced in a sucking baby who teethes early.

Riga's Disease is a chronic inflammatory fibroma or granuloma, which is of frequent occurrence in Central and Southern Italy, and is occasionally met with elsewhere. The Italian peasantry have common names for it, *e.g. Produzione sottolinguale*, but Riga of Naples drew especial attention to the affection in 1881. A white fibrous nodule is seen to be placed astride the fraenum over which the mucous membrane is intact. It is composed of white fibrous tissue containing eosinophil cells. No organisms have been identified. It is due to the rubbing of the fraenum upon a hard gum or precocious incisor whilst sucking, especially when the mouth is not kept clean. The child may be otherwise healthy, or suffer from gastro-enteritis. Usually the malnutrition and the dirty mouth precede the swelling, although the swelling aggravates both conditions until the child ceases to take its food. Inquiries have failed as yet to discover why it should occur sporadically, and why it should be endemic in Italy. Eventually the swelling disappears spontaneously. It may be speedily got rid of by excision, and this may be done when the mouth is clean and the nutrition of the child good enough to ensure primary union of the sutured mucous membrane. After removal of the precocious incisor the swelling has at once disappeared. Usually the treatment is general, and consists in improving the nutrition of the child, keeping its mouth clean, and in avoiding irritation by unsuitable teats. If the swelling shew signs of ulceration it should be painted with a weak antiseptic.

Acute parenchymatous glossitis is especially set up by mercury in an exhausted patient, whether given for syphilis or as calomel. It may also result from infection after a wasp-sting, form part of an attack of erysipelas, or of infection by anthrax or by trichinae. It also follows a chill and exposure to foul odours, especially when alcohol has been freely taken. But glossitis, or *hemiglossitis* when half the tongue is especially swollen, may occur in cases with a dry, foul, neglected mouth, and thus complicate enteric fever and infections generally. The course taken may be that of acute swelling of the tongue and sublingual tissue, with the development of a sublingual abscess; or the oedema may spread to the fauces and produce oedema glottidis, or the absorption of streptococci may lead to septicaemia. In gout and rheumatism the

tongue does not seem to be affected, the pain having its origin in the fauces.

Treatment.—Cleaning the mouth. Scarification of the tongue to relieve oedema. Incision of a sublingual abscess through the floor of the mouth, or beneath the chin. Treatment of oedema glottidis by scarification, intubation, or temporary laryngotomy. Arrest of streptococcal infection by antistreptococcal serum.

Chronic Glossitis.—See under Herpes and Leukokeratosis, pp. 311, 312.

Glossodynia exfoliatiya was described by Kaposi as a condition not due to organisms in which the changes did not spread beyond the epithelium. It depends on the nervous and dyspeptic disturbances noted under Herpes (p. 311). The treatment is the same, local protection by painting with nitrate of silver, careful dieting, the administration of alkalis or alkaline mineral waters and of iron.

Glossitis papillaris is inflammation of the individual papillae, and occurs in gouty and dyspeptic persons, in pulmonary tuberculosis, and in syphilis.

Actinomycosis of the tongue is often but not always started by impaction of a particle of vegetable material in the tongue. An indolent nodule forms, and if it is not excised may become a cold abscess, and after bursting leave a discharging sinus, when a diagnosis may be made from the pus. As the solid nodule cannot be distinguished from tuberculosis, excision is generally adopted as being more speedy and certain; although if the actinomyces granules be noted in the pus, the disease may be cured by increasing doses of iodide of potassium, up to ½ to 1 drm., three times a day.

Tuberculosis of the tongue occasionally starts round a foreign body, and is discovered when the indolent nodule is excised. Tuberculous ulcers of the tongue are generally secondary to pulmonary tuberculosis, and the ulcers are continuously liable to reinfection. They form especially where the teeth have caused an excoriation on the tip or sides. A nodule, a cold abscess, or an indurated fissure may first be seen; or an excoriation may slowly deepen with its base covered with muco-pus and pulpy granulations, whilst the edges are undermined. Rarely the disease is lupus, a slow progressive ulceration which in several cases has been inoculated from pre-existing lupus of the face. The best treatment for an isolated nodule is excision and suture to escape reinfection. If this be not practicable, it is scraped lightly and painted with increasing strengths of lactic acid. Pain may be relieved by orthoform, morphine, or cocaine and starch powder dusted on. But the essential treatment is the general one; unless it is successful, all local measures fail owing to constant reinfection by the sputa.

In *leprosy* there may be pale tubercles or patches on the tongue and palate, which may ulcerate; the tongue may become anaesthetic. In a specimen in the Museum of the Royal College of Surgeons there is a nodular thickening of the dorsal surface of the tongue (*vide* also Vol. II. Part II. p. 663).

Syphilis of the Tongue.—Syphilis has often been inoculated on the tongue by kissing, and among glass-blowers from using the same mouth-pieces. Infection has also, but with less certainty, been attributed to smoking pipes in common, to smoking cigar ends, to passing round wooden cups and spoons, especially in Russia. It has also been traced to the mouthpiece of musical instruments, and has been transferred from one member of a family to others. The situation of the chancre may be determined by excoriations produced by a sharp tooth or tooth-plate. It is nearly always single and near the front of the tongue, at its tip or edge, but it has been seen on the base. A chancre on the tongue forms a raised patch with an excoriated surface, a hard base, and a variable amount of surrounding induration and oedema. Ulceration may give rise to an indurated fissure, or to an ulcer like the bowl of a spoon. A chancre is readily diagnosed by the invariable sequence of indurated and enlarged glands in the neck, and a rash.

Generalised syphilis produces mucous patches which are multiple, especially near the anterior end of the tongue. They consist of hypertrophied papillae with a greyish-white film of mucus and fur. Where the papillae are absent at the edges and under the tongue the patch appears as a mere excoriation, or it may be altered by irritation of the teeth. The diagnosis chiefly rests on finding other evidences of syphilis. They are very infectious. They may be painted with lotio nigra, or bichromate of potash, 10 per cent. When irritated by tobacco smoke or in drinkers the mucous patches may become fissures and ulcers. In adults, especially in smokers and when the teeth are ill-cared for, leukokeratosis follows.

Gummas and sclerosing glossitis start along the middle line of the tongue, or where especially irritated by a tooth. The gumma forms an indolent lump, then breaks down, and when complicated by septic inflammation may, from its surrounding induration and temporary glandular enlargement, be mistaken for cancer. Other evidences of syphilis may suggest a course of treatment, and at any rate a piece from the margin of the ulcer may be examined microscopically before advising an operation.

Tertiary sclerosing glossitis causes an indurated mass, and the affection of the surface by chronic ulcers and fissures, or the epithelium with leukokeratosis, renders the part very liable to cancer,—especially when there is continual irritation from tobacco and alcohol. In all doubtful cases a piece must be removed for microscopical examination.

The *lingual tonsil* is occasionally, very seldom indeed as compared with the other tonsillar structures, affected, either by acute inflammation, so-called lingual quinsy, chronic hypertrophy, or enlargement of the mucous follicles in which may lodge concretions composed of epithelium and leptothrix threads. The veins over the base of the tongue may seem prominent. All that is usually needed is to stop smoking and to improve the patient's health. Local treatment is very rarely required; the exceptional cases are treated similarly to the faucial tonsil.

Thyroglossal tumours are found in the position of the foramen caecum or between it and the hyoid bone, in the line of the thyroglossal tract of His. The swelling has a dusky-red colour and may be covered by intact epithelium; it may vary in size from that of a pea to that of a cherry or more. Blood may become extravasated into a cyst, or the epithelium may yield, and recurrent attacks of haemorrhage occur. They are more common in females, and attention is apt to be drawn to them at the onset of menstruation. The most important point is to make out how much of the thyroid structure is represented in the tongue. The gland in the neck may be entirely absent to palpation, after death a little cystic tissue only having been found in the position of the thyroid. It is therefore wrong to remove the tumour, for, by doing so, most of the thyroid gland tissue the patient has may be removed and the patient may then suffer from myxoedema. The case should first be treated on the same lines as for thyroid hypertrophy, and the swelling will often subside. Only when there is recurrent haemorrhage should the cautery be applied, so as to burn down the projecting portion.

Macroglossia.—Enlargement of the tongue may be present at birth, or a tumour may protrude from the mouth, and has proved removable. They are teratomas or embryomas; dermoid cysts or congenital ranulas may also produce this enlargement. A macroglossia composed of hypertrophied muscular tissue may be seen in idiots; or the hypertrophy may be unilateral, and coincide with muscular hypertrophy of one half of the body. Lymphangiomatous macroglossia may start from a circumscribed lymphangioma of the tongue, or be from the first diffuse, the surface of the tongue being thickened like that of a calf, lingua vituli.

A peculiar enlargement of the tongue has been found to be due to neuro-fibromatous enlargements of the lingual or hypoglossal nerve. Other manifestations of von Recklinghausen's disease may be present, but the condition of the tongue has existed alone in the case of children.

Malignant disease of the tongue is mainly of surgical interest (see References under Leukokeratosis, p 327).

For Nervous Affections of the Tongue see references 1, 171, 172, 173, 174.

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DISEASES OF THE OESOPHAGUS

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Methods of Examining the Oesophagus.—By external examination enlarged cervical glands, secondary to carcinoma of the oesophagus, may be felt; and their discovery would help to clear up any doubt as to the cause of dysphagia. A pharyngeal pouch may be felt to swell up when food is taken, and can be emptied by pressure applied externally.

Internal examination gives information as to the presence and situation of a stricture or foreign body.

The use and method of employing the oesophagoscope will be described in the volume on Diseases of the Throat and Ear (Vol. IV. Part II.), by Mr. Waggett. Other means at our disposal are:—

(i.) *The Passage of an Oesophageal Tube or Bougie.*—The pharynx may first with advantage be sprayed with a 10 p.c. solution of cocaine. The bougie should be warmed and lubricated with glycerin, not with oil, which is very repugnant to the patient. The patient sits in a chair and the bougie is passed along the posterior wall of the pharynx into the oesophagus. The bougie is very likely to be arrested as it passes into the oesophagus behind the cricoid cartilage, about six inches distant from the incisor teeth, even when no morbid condition is present; the patient should then be directed to swallow, in order to help the onward passage of the instrument. The point at which any obstruction is encountered should be noted by measuring the distance from the incisor teeth. The distance from the incisor teeth to the stomach is at least 15½ inches, and may be even more. By means of the bougie, spasm, stricture due to organic causes, and paralysis can be distinguished from each other. If, in the case of stricture, a bougie with a small depression near the end is passed, some of the growth, if it be a case of carcinoma, may be removed and examined microscopically and the diagnosis settled.

(ii.) *Auscultation* of the oesophagus was first practised by Hamburger, and was described in this country by Mackenzie and Professor Clifford

Allbutt. The oesophagus is auscultated from without while the patient swallows water or gruel. In order to employ this method the observer must make himself familiar with the characters of the normal oesophageal sound. The rapidity, or rate at which the morsel is swallowed, can be timed by placing the hand on the hyoid bone. In organic stricture the sound of the passage of the morsel is much altered, prolonged, or transformed into a continuous and confused noise; in spasmodic stricture the sound is unaltered; in paralysis of the gullet it is absent.

Skiagrams taken by means of the *x*-rays demonstrate the presence and position in the oesophagus of foreign bodies, diverticula, and dilatation.

MALFORMATION.—*Congenital Atresia.*—In this rare condition the oesophagus is divided into two parts. The upper segment, continuous with the pharynx, ends blindly a short way above the level of the bifurcation of the trachea. The lower segment opens in a normal manner into the stomach, but takes its origin from the trachea above the bifurcation, or in rare instances from a bronchus. At first sight this condition might be regarded as analogous to that of imperforate anus, and to be due to a failure of union between the stomodaeum and the foregut. But this explanation is incompatible with the development of the pharynx from the foregut. This atresia is in reality due to a secondary obliteration of the oesophagus (Shattock), occurring opposite the diverticulum from which the lower part of the trachea and the lungs are developed, and depends on malformation of the tracheo-oesophageal septum. In a few cases associated malformations have been recorded; these have been collected by Dr. Ballantyne. Infants presenting this malformation are born well nourished, but of course are unable to swallow, and the amount of fluid vomited is the same as that taken. They die of starvation about the fifth day after birth. The malformation can be detected by passing a bougie. No treatment has been successful, but gastrostomy, though not very hopeful, is indicated. More extreme degrees of malformation have been met with in monsters.

Congenital stenosis of the lower end of the oesophagus is very rare, but a few undoubted cases are on record. It usually occurs just above the diaphragm.

The *persistence of branchial clefts* may lead to a fistulous communication between the pharynx and the exterior. The external opening is situated along the anterior border of the sterno-mastoid muscle, and is often associated with the presence of an accessory auricle. Either the internal or the external orifice may be closed. If both are obliterated the intervening portion may undergo cystic dilatation; the contents of these branchial cysts are either mucoid or sebaceous in character. The multilocular serous cysts of the neck (hygroma) are cystic lymphangiomas, and are not derived from the branchial clefts. In rare instances carcinoma may arise in these branchial cysts. The branchial fistulas or pouches are, it is to be noted, lateral in position, and communicate with the pharynx, not with the oesophagus.

(1) *Diverticula of the oesophagus* are pouch-like protrusions of part of the wall of the gullet, and must be distinguished from dilatation of the whole circumference of the oesophagus. There are three varieties: (i.) Pressure or pulsion, (ii.) Traction, and (iii.) Traction-pressure diverticula.

(i.) *Pressure Pouches.—Syn.*: Oesophageal, pharyngeal pouches, pharyngoceles, pulsion diverticula, Zenker's diverticula. Pressure pouches, though often called oesophageal, in reality arise from the lower part of the pharynx, or from its junction with the oesophagus. This is, however, a convenient place to describe them.

A pressure pouch begins in the posterior median line as a small depression which gradually becomes larger from the lodgment of food in it. A diverticulum is thus formed between the oesophagus and the spine, which points downwards; and, from the inclination of the oesophagus to the left, often projects on that side of the neck; though it may be present on both sides, or even pass into the posterior mediastinum (Pollard). As the pouch gets larger it becomes the direct continuation of the pharynx, more and more food enters it, and swallowing becomes increasingly difficult. Structurally the pouch is composed of thickened mucous membrane and connective tissue. The neck of the sac is enclosed by muscular fibres derived from the inferior constrictor muscle; but although the pouches may look as if they had a muscular wall, microscopic examination often shews that this is not the case. Their wall, however, is as thick as that of the oesophagus. They vary very considerably in size, and may contain as much as nine ounces. They are elastic and can be distended to twice or three times their ordinary size. Their average length is four to five inches. In very rare cases squamous-celled carcinoma has developed in a pressure pouch.

These pressure pouches are, as the name implies, caused by pressure exerted on the wall of the tube from within. Their constant situation depends on the anatomical structure and arrangement existing at the junction of the pharynx and the oesophagus. In this situation the lumen of the passage narrows considerably, the anterior wall, being formed by the cricoid cartilage, is firm and unyielding, while the posterior muscular wall at this point is thinner and weaker than in any other part of the oesophagus. This condition is, of course, congenital; but there is nothing to suggest that the pouches themselves are congenital in origin. A hard bolus of food being driven against this place of less resistance might produce a hernial protrusion of the mucous membrane which, as time went on, would become progressively larger. Thus, traumatism may be the immediate cause of such a pouch; but the situation is determined by the normal anatomy of the part. Stricture of the upper part of the oesophagus and pressure exerted from without by tumours, such as goitres, have been supposed to act as causal agents. Pressure pouches are rare, but when they do occur they are met with most often in patients past middle life or even in old age.

Though symptoms are not known to have been present in all the specimens, in most of the recorded cases they have been well marked. The usual course of the disease is as follows:—

First, there is return of undigested food some time after a meal, perhaps with coughing, and the sensation of a foreign body in the throat. This regurgitation increases in amount, and is after a time associated with distinct difficulty in swallowing. The breath may be offensive from decomposition of fragments of food in the pouch. As the pouch increases in size the swelling of the neck after eating or drinking becomes more noticeable. The pouch can be emptied by pressure applied from without. Swallowing gradually becomes very difficult, and a probang which previously could be passed down the oesophagus now enters the pouch. After giving bismuth mixed with food the presence of the pouch can be shewn by the *x*-rays. Finally, unless relieved, the patient dies of slow starvation.

Treatment.—Removal of the pouch has given brilliant results; and is the radical and ideal treatment. Of 23 cases collected by Veiel 18 recovered; in 42 collected by Zesas 8 died from the operation, in the remainder a fistula formed in all but 6 cases, which closed in from four days to sixteen weeks. Life has been prolonged by gastrostomy; but in these cases the cause of the dysphagia was not correctly diagnosed till after death.

(ii.) *Traction diverticula* generally occur on the anterior wall of the oesophagus near the bifurcation of the trachea, and are commonly due to adhesions between inflamed bronchial glands and the wall of the oesophagus. As the glands and adhesions contract, the wall of the oesophagus at this spot is pulled out, and a pouch results. Traction diverticula may occur elsewhere, as the result of the contraction exerted by pleural or pericardial adhesions. They are never more than an inch deep. Uncomplicated traction diverticula do not give rise to any symptoms. In rare instances a sharp foreign body, such as a piece of bone, may perforate the wall of the pouch with serious results. Suppuration may occur, or the superior vena cava, pulmonary artery, or aorta may be penetrated. Ulceration of the wall of the diverticulum may lead to a tracheo-oesophageal or broncho-oesophageal fistula, and so to septic bronchopneumonia and gangrene of the lung. In a similar way, pleurisy, pericarditis, or a mediastinal abscess may be set up.

(iii.) *Traction-pressure* diverticula. In this form a traction diverticulum becomes enlarged and distended by the entrance of food and gives rise to symptoms resembling those of a pressure pouch. The fundus of the pouch may rest on the diaphragm. They are extremely rare.

Dilatation of the Oesophagus.—Contrary to what might have been expected, and indeed is often stated, dilatation above a stricture of the oesophagus is far from common. In most cases food, if unsuccessful in passing through the stricture into the stomach, does not accumulate to any extent above the obstruction, but is rejected shortly after being

taken. When dilatation secondary to a stricture does occur, the point of obstruction is usually near the diaphragm.

In *primary, idiopathic,* or *diffuse dilatation,* wherein there is no manifest mechanical cause to account for the condition, the whole of the oesophagus may be affected; but the lower two-thirds or the middle of the tube are most dilated, so that the oesophagus may have a spindle-shaped outline tapering towards its two ends; it may be six inches or more in circumference. At the point where it passes through the diaphragm the oesophagus is usually of its normal size and presents no sign of organic stricture. The dilated oesophagus, which may have a capacity of a pint or more, may become lengthened, and by bending on itself present a tortuous outline. The muscular walls are usually hypertrophied, and the mucous membrane may be normal, thickened, inflamed, or ulcerated.

Etiology.—The cause of this somewhat rare condition—primary dilatation—has given rise to considerable discussion. Morell Mackenzie considered that it was due to atony, either congenital or acquired, or to weakness of the oesophageal wall in its whole circumference.

It is remarkable that, in most cases of simple dilatation, the muscular coat of the oesophagus is hypertrophied. Dilatation with wasting and thinning would be a natural result of muscular weakening; but dilatation with hypertrophy forcibly suggests some obstruction at the cardiac end, and if no organic stenosis be present it is possible that some functional obstruction existed during life. This could be explained by continued closure of the cardiac orifice of the oesophagus, which might conceivably be due to a spasm of the sphincter fibres at the lower end of the circular coat of the oesophagus—cardiospasm; or to paralysis of the longitudinal muscular fibres, by the contraction of which the cardiac orifice of the stomach is opened. The generally accepted opinion is that the dilatation is due to cardiospasm. Continued spasm might be expected to lead to marked local hypertrophy of the circular muscular coat near the cardia; this condition appears to have been present in some but not in all the cases recorded.

By the alternative view paralysis of the longitudinal muscle would explain dilatation; by leaving the cardiac orifice closed it would lead to obstruction at the lower end of the oesophagus, and thus might induce general hypertrophy of the circular coat. Simple dilatation with hypertrophy, then, can perhaps be explained more satisfactorily as the result of inhibition and atony of the external longitudinal coat than as due to weakness of the whole of the muscular wall. Prof. Langley has shewn that the vagus in the cat contains fibres which inhibit the sphincter at the cardia, and degeneration of the vagus has been described in one case by Kraus, but was not present in a case examined at St. George's Hospital (Lazarus-Barlow). At present this explanation cannot be said to be in any way proved. We can only say that dilatation of the oesophagus, like the same condition in other hollow muscles—such as the heart, bladder, stomach, intestines—is usually accompanied by hypertrophy; but the hypertrophy, especially in the thinner-walled viscera,

usually fails to compensate or to correct the dilatation. It must be remembered that what appears to be a local dilatation of the oesophagus immediately above the diaphragm may be part of the fundus of the stomach drawn through the diaphragm in cases of advanced visceroptosis (Keith). Localised dilatation of the oesophagus above the cardia has been noted in merycism or habitual rumination.

Symptoms.—The onset is usually gradual, but it may come on rapidly. There is regurgitation of food in an undigested state, except for some transformation of starch into sugar, with much mucus, some hours after eating. The breath is usually foul, and there is often salivation. Pain of a burning character may be felt in the gullet. The disease is slow in progress and need not necessarily shorten life. The condition can be recognised by oesophagoscopy, and by the *x*-rays after the administration of bismuth. Diagnosis from a traction-pressure diverticulum arising in connexion with the lower half of the oesophagus is difficult unless Rumpel's differential test be employed.

Treatment.—In the early stages of cardiospasm local application of a solution of 3 to 8 p.c. solution of silver nitrate or cocaine may be tried; but when dilatation has supervened mechanical treatment of the obstruction should be employed. The passage of bougies has not been followed by relief; Sippy has obtained considerable success—nine cures—with a rubber-bag dilator, previously employed by Mr. Lockwood, by means of which it is possible to obtain any degree of dilatation without an anaesthetic. Goldmann advocates forcible dilatation of the cardia by Mikulicz's method, and refers to six cases successfully treated in this way. The treatment of dilatation secondary to stricture is of course that of the primary affection.

Hypertrophy of the oesophagus is occasionally found after death apart from any organic stenosis of its lumen; thus, it may occur in pyloric obstruction due to carcinoma or in congenital hypertrophy of the pylorus. In connexion with a case which I recorded in 1898 the view was put forward that apparently idiopathic hypertrophy of the oesophagus is an early stage of dilatation.

Oesophagitis. — **Thrush** or parasitic stomatitis, due to a fungus *Blastomyces* or *Oidium albicans*, may spread from the mouth and pharynx of infants to the oesophagus (*vide* p. 306). Thrush may also occur in adults in the last stages of wasting diseases, such as pulmonary tuberculosis. It may attack the whole length of the gullet, or but a part of it; the lower half of the tube near the stomach is more often affected. It may be discrete or confluent, and by exuberant growth may actually obstruct the lumen, and so interfere with swallowing.

There may be no symptoms to suggest that the oesophagus is affected; or, on the other hand, swallowing may be difficult or even impossible.

Diagnosis.—Its presence may be suspected when the above symptoms arise in a case of thrush in the mouth; but otherwise it can hardly be recognised.

The *treatment* is much the same as that of thrush in the mouth; namely, attention to cleanliness in feeding, and borax or similar agents given by the mouth.

Diphtheria. — Diphtheritic inflammation occasionally spreads from the fauces into the oesophagus; Councilman, Mallory, and Pearce found this in 5 per cent of 220 autopsies; and in very rare cases membrane has been found to extend even into the stomach. It gives rise, however, to no characteristic symptoms, except that the cast of the tube may be brought up.

Non-diphtheritic membranous oesophagitis is very rare; it has been said to occur in association with various febrile disorders. Some cases, such as that ascribed by Dr. Nathan Raw to drinking neat spirit, in which a cast of the oesophagus was brought up, may be minor degrees of traumatic oesophagitis (*vide* p. 337).

Simple Acute Oesophagitis. — *Acute Catarrhal Oesophagitis* is rarely seen, and its etiology is equally obscure. It may spread from the pharynx, and has been attributed to the local action of alcoholic drinks, to hot food, to cold applied either internally, as in eating ices or drinking iced water, or externally. It has also been attributed to rheumatism. It is said to occur sometimes in the course of the specific fevers, and to account in some degree for the anorexia met with in them. Extension of inflammation from adjacent parts—for example, in pericarditis, pneumonia, or pleurisy—is seldom manifested clinically by pain or difficulty in swallowing. Post-mortem examination, however, often shews that the inflammation has spread at any rate to the muscular walls of the oesophagus. Lastly, acute oesophagitis may arise without any assignable cause, especially in sucklings. The mucous membrane is injected, swollen, may shew submucous oedema, and is covered with mucus.

Phlegmonous inflammation of the oesophagus is a rare and very severe affection. It may follow the acute catarrhal form, or the impaction of foreign bodies. The spread of suppuration from without, from an ulcer in the wall of the tube, or even from phlegmonous gastritis, may give rise to it. The mucous coat may be separated or dissected off by diffuse suppuration occurring in the submucous coat. Ulceration or even gangrene (gangrenous oesophagitis) may result. It may prove fatal in a few days; if recovery take place, cicatricial contraction is likely to follow and to lead to stricture.

Symptoms.—In severe cases there is extreme pain on swallowing even liquid food, so much so that the patient hardly dare try to relieve the urgent thirst which characterises the affection, or to swallow his saliva. When an attempt is made there is very considerable difficulty due either to reflex spasm or paralysis. Speaking and movement are painful, there is tenderness on pressure, and complaint may be made of a dull aching pain in the neck. Glairy mucus is expectorated which, in phlegmonous oesophagitis, may contain pus and blood.

Treatment in severe cases consists in absolute rest to the oesophagus,

the patient being fed by enemas. Pain and discomfort may be relieved by morphia and by the external application of poultices and hot fomentations. Adrenalin chloride has been found to exert a beneficial local effect on the oesophageal mucosa. In milder forms demulcent drinks and ice by the mouth may be given, and any concomitant pharyngitis or gastric disturbance must be appropriately treated.

Traumatic Oesophagitis.—Acute traumatic oesophagitis may be due to swallowing very hot fluids—such as boiling water, or inhaling steam from the spout of a kettle. Children are especially apt to injure themselves in this way. But most frequently it is the result of irritant or corrosive poisons, taken by accident or with suicidal intent. Strong solutions of acids and alkalis produce necrosis of the mucous membrane, which may be expelled as a complete cast; and occasionally gangrenous oesophagitis may result. Weaker solutions give rise to varying degrees of inflammation and ulceration. The mucous membrane of the oesophagus may be affected in parts only, and is often much less damaged than that of the stomach.

Symptoms.—Immediately on taking the poison there is a burning pain in the gullet extending to the stomach, and great pain on attempting to swallow. The symptoms may be somewhat masked at first by collapse; but as the patient rallies he becomes characteristically anxious. Glairy mucus stained with blood may be brought up. The lips, tongue, and mouth will shew the effects of the poison; unless, as sometimes happens in suicides, the poison were carefully poured by means of a long-necked bottle into the pharynx. The dysphagia and pain continue, and the symptoms resemble those of acute catarrhal oesophagitis; though they are of course more marked in degree.

Treatment.—If the poison taken was an acid, it should be counteracted by dilute alkaline solutions, which may be given by a small tube passed by way of the nose half-way down the oesophagus, but not by the stomach-pump. Injury by alkalis should be treated, not by the administration of mineral acids, but of dilute vinegar or oil.

Liquid food only should be given by the mouth, and the patient's strength maintained by nutrient enemas.

After-Results.—In severe cases the difficulty of swallowing, due to the inflammation and ulceration, passes uninterruptedly into that due to the resulting cicatricial contraction. In milder cases there is an interval between the initial dysphagia and that due to cicatricial stenosis; stricture usually makes itself manifest within a year after the occurrence of traumatic oesophagitis.

Chronic Oesophagitis.—Chronic inflammation of the oesophagus may accompany other diseases of the tube, such as carcinoma or stricture; and may be associated with syphilis or pulmonary tuberculosis (Mackenzie); but it is rarely seen post-mortem, and, generally speaking, is not recognised clinically. It is probable that in many cases of chronic gastritis, especially when there is pharyngitis as well, there is also some chronic oesophagitis; but the manifestations of the former overshadow

any that may be due to changes in the oesophagus. The relationship between chronic alcoholism and chronic oesophagitis is doubtful.

In chronic heart disease, and in other cases in which the oesophagus has been exposed to external pressure—as, for example, from a mediastinal growth—chronic oesophagitis may be met with.

Morbid Anatomy.—There is thickening and opacity, general or patchy, of the mucous membrane, which is covered by tenacious mucus. In places there are small elevations or warts composed of thickened epithelium covering hypertrophied papillae. These warts are of no clinical importance.

In sprue, the oesophagus is denuded of epithelium, and shews extensive inflammatory changes in the underlying coats. There is considerable pain on swallowing, as might be expected. The chronic oesophagitis here is of quite a different nature from that just described. (*Vide* art. on "Sprue," Vol. II. Part II. p. 545.)

Chronic oesophagitis may, it is thought, lead to ulceration, and thus by perforation to peri-oesophageal abscess; or it may dispose to rupture. Cicatrisation of such ulcers or submucous fibrosis may explain some cases of oesophageal stricture of otherwise obscure origin.

Non-malignant ulceration is somewhat infrequent. It may be due to various causes; the most important forms are those due to the impaction of foreign bodies, *e.g.* tooth-plates, and to swallowing corrosive fluids (*vide* p. 337). In poisoning by tartar emetic, multiple superficial ulcers may result. Ulceration of the mucous membrane over dilated oesophageal veins near the cardiac end is important as the cause of severe haematemesis, especially in hepatic cirrhosis. Exogenous ulceration of the oesophagus by a thoracic aneurysm need not be considered under this heading. Decubital ulceration occurs in patients debilitated by exhausting disease, such as pulmonary tuberculosis or enteric fever, at the upper end of the oesophagus, and is due to the pressure exerted by the cricoid cartilage; its presence is very rarely suspected in life. In new-born infants ulceration may follow idiopathic oesophagitis, and occur either at the upper or lower end. Catarrhal ulcers are superficial erosions in catarrhal oesophagitis. Syphilitic ulceration does occur, but is very rare; its existence is often assumed in order to explain stenosis in a syphilitic subject. In one case in which ulceration discovered at the autopsy was at first thought to be syphilitic, subsequent microscopic examination proved it to be a rapidly growing squamous-celled carcinoma. Tuberculous ulceration is very rare, and of no clinical importance. It may be due to extension from the bronchial glands, to infection of simple ulcers by sputum, to perforation of tuberculous abscesses, extension from the pharynx, or be part of generalised tuberculosis. Ulceration has been described in a number of acute infectious diseases, but it is undoubtedly rare. In enteric fever ulcers other than the decubital mentioned above are very infrequent: Louis, however, recorded 7 examples; Mitchell, who analysed the cases at the Johns Hopkins Hospital, only met with one example. In about 6 cases (Tinker,

Dennis) stricture has followed. Ulceration has also been recorded in scarlet fever, pneumonia, pemphigus, small-pox. Simple ulcers in the lower part of the oesophagus, resembling gastric ulcers in their appearance, have been called peptic ulcers, and are regarded as due to the action of the gastric contents. They occur in cases in which vomiting has been present, and so may be associated with chronic dilatation of the stomach. Tileston, who has collected 36 cases examined after death, finds that they are usually single and confined to the oesophagus, that in a few instances the ulceration extends from the cardiac end of the stomach, and that in some cases there is associated ulceration in the stomach or in the duodenum. They occur more often in men, and in middle life. It must be remembered, however, that destruction of the mucosa of the lower end of the oesophagus may be due to post-mortem digestion.

Symptoms.—In many cases ulceration is entirely latent and is only discovered after death. In some instances the first manifestations are those due to perforation or rupture. The symptoms when present are most commonly pain on swallowing, usually referred to the xiphoid cartilage, accompanied by dysphagia due to spasm set up by the irritation of the ulcer, and haemorrhage which occurred in 53 per cent of Tileston's cases in which a clinical history was available. The diagnosis of oesophageal ulcers, except by the oesophagoscope, is very difficult.

Treatment should be that of complete rest to the oesophagus, the patient being provided with water and nutrient enemas; subsequently liquid and pulpy food should be given by the mouth. Some benefit may be expected from the local action on the ulcer of nitrate of silver and bismuth. The introduction of bougies should, of course, be avoided.

Results of Simple Ulceration.—Healing of a simple ulcer may give rise to cicatricial stricture of the oesophagus.

The presence of simple ulceration disposes to rupture of the oesophagus, and has probably often preceded it. Perforation into the pleura may set up pneumothorax or a pyopneumothorax; or a fistulous communication with the bronchi may result. Probably many of the cases of tracheo-oesophageal or broncho-oesophageal fistula, not of malignant origin, are due to softening of tuberculous bronchial glands putting the two tubes into communication; others are due to a perforating ulcer in the oesophagus.

Rupture.—Rupture is a very rare accident; so much so that some reported cases have been regarded as only post-mortem digestion of the walls of the oesophagus from regurgitation of the contents of the stomach. But there is no doubt that the oesophagus has been ruptured as the result of violent vomiting, retching, or straining. At least 30 reputed cases are on record.

Etiology.—It is probable that there is usually some local alteration in the wall of the oesophagus; either that it was weakened by ulceration at the spot where the rupture eventually occurred, or that there was

some narrowing or obstruction, as, for instance, by a foreign body, above the point of rupture; so that when the contents of a full stomach were driven up into the oesophagus very forcible distension took place. Rupture has been produced by the vomiting set up by an anaesthetic given for the removal of sharp spicules of bone impacted in the oesophagus. The rupture always occurs in the lower part of the oesophagus, which Mackenzie has shewn to be the weakest part, and is in the long axis of the tube. Nearly all the victims are males.

Symptoms.—Agonising pain comes on directly after the rupture has presumably occurred, and is increased on movement. Though the patient is able to swallow yet, as the food passes into the pleura, he can no longer vomit it from the stomach; but he may bring up a little blood. Marked collapse supervenes, and death follows after an interval varying from a few hours to a few days. So far as we know, it is invariably fatal. Subcutaneous emphysema has been noted in some cases as a result of rupture of the oesophagus.

The *diagnosis* is very difficult. If rupture be suspected, food should be given either by rectum or by means of a Symonds' tube kept permanently in the stomach (*vide* p. 348).

Stricture of the Oesophagus.—The oesophagus may be pressed upon from without; stricture thus brought about may be spoken of as *extrinsic*. Thus, in the neck tumours in connexion with the thyroid body may lead to dysphagia; while in the thorax a mediastinal new growth, dislocation backwards of the sternal end of the clavicle, abscess in connexion with Pott's disease of the spine, aneurysm of the aorta, or, very rarely, a large pericardial effusion, may compress and narrow the lumen of the oesophagus. But it is remarkable how comparatively tolerant the oesophagus is of pressure exerted from without.

Intrinsic stricture or narrowing due to some change in the walls of the oesophagus is of the following kinds:—

(i.) Spasmodic stricture; described under neuroses of the oesophagus (p. 350).

(ii.) Simple stricture: this is a rare condition, and its etiology is somewhat doubtful. When it occurs the stricture is found in one of two situations: (*a*) near the stomach, or (*b*) some little distance below the cricoid cartilage. The stricture shews no evidence of former inflammation, and there is no formation of cicatricial tissue. The stricture is membranous, and consists of a fold of mucous membrane projecting inwards towards the middle of the lumen of the tube, like a diaphragm. It has been thought to be congenital; and very possibly some of the cases occurring in the upper part of the oesophagus are thus explained, and are due to an arrested stage of the kinking process which, when fully developed, gives rise to congenital atresia of the oesophagus.

Against this it may be urged that the symptoms in most cases are manifested in middle or even in advanced life, and were not marked at an early age. Sir W. Kendal Franks suggests that such a stricture is the result of spasmodic contraction of the muscularis mucosae, or

circular muscular coat; possibly reflex in origin. Congenital stenosis of the oesophagus is referred to on p. 331.

Fibrous or Cicatricial Stricture of the Oesophagus.—The healing of an ulcer may, by subsequent contraction, lead to progressive and permanent narrowing of the lumen — a process analogous to the production of a urethral stricture. Thus, it may follow ulceration set up by the impaction of foreign bodies, or by rough or sharp pieces of food. A gastric ulcer in a somewhat unusual position round the cardiac orifice may lead to oesophageal obstruction. In syphilis the contraction of cicatrices, or the presence of a gumma in the wall of the tube, might give rise to dysphagia; but it is very rare indeed to find evidence post-mortem of syphilitic change in the oesophagus. On the other hand dysphagia in syphilitic patients may yield to treatment with iodides and mercury, and so justify the diagnosis of syphilitic stricture. Stenosis due to actinomycosis has been recorded (Marchant, Garde). It has been thought that chronic oesophagitis may, by ulceration and subsequent organisation, account for some cases of fibrous stricture.

But the commonest cause of fibrous stricture is undoubtedly inflammation, ulceration, and subsequent cicatrisation of the mucous and underlying coats, due to the action of acids, alkalis, hot fluids, and the like. When a strong solution of a corrosive poison has been taken, the primary difficulty (dysphagia) and pain (odynphagia) in swallowing pass almost uninterruptedly into the dysphagia caused by the cicatricial narrowing of the lumen of the gullet. When a weak solution has been taken, the pain and difficulty in swallowing due to the ulceration pass away as the surface heals; but in a few months dysphagia recurs, and, as the cicatrisation progresses, becomes more marked.

The stricture may be single and annular, or of considerable extent, 2 to 3 inches or more; or there may be several strictures. Above the obstruction the muscular walls may be hypertrophied, and occasionally the tube may be dilated.

Inflammation around the stricture may give rise to a peri-oesophageal abscess; or the perforation of an ulcer to pleurisy, gangrene of the lung, or an abscess in the immediate neighbourhood of the gullet.

Symptoms.—The dysphagia begins gradually, and is progressive and permanent. Inability to take solid food may come on suddenly, and is succeeded by dysphagia for liquids. From added spasm the obstruction may vary in degree, but never entirely disappears as it does in pure spasmodic stricture. A bougie is always arrested at the same point in its progress; and by means of auscultation the situation of the stricture can be confirmed. Food is returned shortly after being taken, and is alkaline in reaction. The food generally seems to the patient to stop at or about the episternal notch. Emaciation is a natural result of the chronic starvation.

Diagnosis.—The history that an irritant or corrosive poison has been swallowed is an important factor. In cases of syphilis other signs of the disease, and the relief of the symptoms after antisyphilitic treatment,

point to a stenosis of specific origin; but the diagnosis of syphilitic stricture can never amount to more than a conjecture (Mackenzie). When there is no history of an injury, an aneurysm should always be first suspected: then carcinoma, and, lastly, simple stricture. If there be the least suspicion of an aneurysm a bougie must not be used; and in malignant stricture great care should be taken, as perforation may be easily produced. The stricture can be seen by an oesophagoscope.

The early symptoms of traumatic and malignant stricture are indistinguishable; but the history of traumatic oesophagitis, and the slow progress of the dysphagia, distinguish the former from the stenosis due to carcinoma. Signs of pressure on the recurrent laryngeal nerves should suggest aneurysm or malignant stricture; pressure on these nerves, in innocent strictures of the gullet, is rare. Carcinoma, however, may arise in a fibrous stricture of some standing.

Several methods of treatment have been advocated and practised; gradual and rapid dilatation, internal oesophagotomy, opening the oesophagus below the stricture or external oesophagotomy, the introduction of a Symonds' tube, and gastrostomy. Rapid dilatation and internal and external oesophagotomy are uncertain in their effects and dangerous, and should not be employed. Gradual dilatation should be first employed, oesophageal bougies of increasing size being passed every other day or so. The bougie is left for some minutes in the stricture, and then withdrawn; the same sized bougie should be introduced several times before proceeding to the next size. When dilatation is complete a bougie should still be passed at intervals of a month, to prevent relapse. Electrolysis has been employed with success in some cases.

In cases in which the diagnosis between cicatricial and malignant stenosis is doubtful, a Symonds' tube should be worn (*vide* p. 348). If the stricture have become impassable, and gradual dilatation or the introduction of a Symonds' tube be therefore impossible, gastrostomy must be performed without delay. Life may be sustained for some time by rectal feeding, and for this purpose Leube's solid enema is the best; but the operation of gastrostomy should not be postponed until the patient is moribund (*vide* treatment of Malignant Stricture, p. 348).

In the rare condition of simple or membranous stricture Sir Kendal Franks has performed oesophagectomy with success.

Prognosis.—If the patient come under observation early, so that the stricture can be treated and his nutrition kept up by food, the outlook is good. If, on the other hand, the narrowing has become extreme and the patient is emaciated, there is danger that he may die of exhaustion before treatment—usually gastrostomy—has had time to work any good.

This form of stricture is compatible, under treatment, with a very considerable lease of life.

Malignant Stricture of the Oesophagus.—Fourteen cases of *sarcoma* of the oesophagus have been collected by Messrs. Corner and Fairbank. In 3 cases the tumours were pedunculated. The age incidence and symptoms are much the same as in carcinoma; but the pain is said to be

more severe and the course more rapid in sarcoma. Sarcoma of the lymphatic glands in the immediate neighbourhood of the oesophagus may lead to stenosis; but this is not primary sarcoma of the gullet, and is a much commoner event. Sarcoma of the oesophagus may be a mixed-, round-, or spindle-celled growth.

Etiology.—Carcinoma of the oesophagus occurs much more commonly in men than in women. In 820 cases, obtained by adding together the statistics given by Mr. Newman (510 cases), the Middlesex (225 cases) and St. George's (85 cases) Hospitals, 672 or 82 per cent were in men, and 148 or 18 per cent in women.

In 7297 cases of primary cancer of various parts of the body, collected from the records of four large metropolitan hospitals by Mr. Roger Williams, 2669 were in men; and of these, 144 were primary in the oesophagus—a percentage of 5·3; while in the 4628 cases of cancer in women the oesophagus was primarily affected in 35, or 0·7 per cent. In 4732 cases of cancer from the Middlesex Hospital, tabulated by Dr. Lazarus-Barlow, in which the percentages were slightly lower, the cases of primary carcinoma of the oesophagus were rather less than half those of primary gastric cancer. In another series the oesophagus was affected in 58 and the stomach in 123 (Mathieu and Debrovici).

A family history of malignant disease has been supposed to indicate a proclivity to carcinoma of the oesophagus. Mackenzie found such a history 11 times in 60 cases, and quotes 10 cases, recorded by Richardson, in all of which it was present. In the 85 cases from St. George's Hospital a family history of malignant disease is mentioned in 7 only; in one a brother of the patient died in the hospital of carcinoma.

Alcoholism and long-continued dyspepsia have been suggested as causes of carcinoma; but there seems little to support this opinion. It has been suggested that enlarged bronchial glands may by irritation be a causal factor in the development of carcinoma of the gullet opposite the bifurcation of the trachea. It is certainly remarkable that carcinoma frequently occurs in the parts of the oesophagus which are normally narrower than the rest, and thus more subject to irritation. Tileston refers to 2 cases of carcinoma arising on the scar of an old ulcer.

Influence of Age.—The disease is rare before 40, and commonest about 55. Dr. V. D. Harris recorded a very exceptional case in a man aged 21.

Women are attacked at an earlier age than men: thus, in 100 cases collected by Mackenzie, all those under the age of 40—8 in number—were in women. The average age in woman is about 50; in man it is about 55.

Situation of the Growth.—Considerable discussion has taken place as to the commonest site for carcinoma in the oesophagus; the upper, middle, and lower thirds being each thought by various observers to be the part most often attacked. Mr. Butlin points out that if the oesophagus be divided into halves instead of into thirds, the number of times each half is affected is much the same. This is fairly borne out by the cases from the post-mortem records of St. George's Hospital, as follows:—

Upper third . .	18	Upper half . . .	28
Middle „ . . .	19	Lower „ . . .	31
Lower „ . . .	22		

In 445 cases collected by Mr. Newman, the upper third was, however, more often affected than the other two put together; his figures are:—Upper third, 227, or 51 per cent; middle third, 98, or 22 per cent; lower third, 120, or 27 per cent. As this author points out, though necropsy reveals the extent of the growth, it does not necessarily shew the point where it began; for it may spread considerably during the patient's life. When the growth is at the junction of the oesophagus and pharynx there may be some difficulty in determining where it began. This, together with the somewhat arbitrary line of demarcation between these continuous channels, may account for some of the differences in the statistics. In the cases collected by Dr. Nothwanger from the records of St. George's Hospital, it is noteworthy that 8 out of the 10 cases of carcinoma in women were in the upper third of the tube.

The growth may spread either from the oesophagus to the pharynx or conversely. Carcinoma at the lower end of the oesophagus may spread to the cardiac orifice of the stomach, and conversely; in two cases of extensive carcinoma of the cardiac end of the stomach I have seen the growth spreading into the submucosa of the oesophagus. The microscopic characters of the growths were incompatible with the view that the growth had spread downwards from the oesophagus. Carcinoma of the cardiac orifice, as distinguished from carcinoma of the cardiac end of the stomach, was regarded by Hilton Fagge as belonging to the oesophagus and not to the stomach; this, however, has been disputed by Perry and Shaw (*vide* p. 346).

Morbid Anatomy.—Carcinoma beginning in the epithelial lining passes into the loose submucosa, and tends to spread transversely round the lumen of the tube; while at the same time it infiltrates the muscular coats. The extent to which it invades the circumference of the oesophagus varies; but frequently, as in other parts of the alimentary canal, carcinoma produces an annular stricture. Obstruction depends chiefly on this annular character of the growth, but also in some instances on the projection of a nodular, or even occasionally polypoid, growth into the lumen of the tube. It is remarkable how rare dilatation is above a malignant stricture. There may be no hypertrophy of the walls even above a tight stricture. At some distance from the growth small white nodules of a similar structure may occur in the mucous membrane. Mr. Targett has recorded a case in which a fibrous polypus of the oesophagus, lying 4 inches above a carcinomatous growth in the oesophagus, became invaded superficially by the growth; possibly by implantation effected by catheterisation. In rare instances there is another growth in the stomach, possibly due to implantation.

Ulceration begins early; it was absent in 3 cases only out of 55 collected from the records of St. George's Hospital. Ulceration of the

growth diminishes the obstruction ; and, if very early or rapid, may explain the occasional latent cases of oesophageal carcinoma without any dysphagia. Ulcerated particles of growth may in rare cases be vomited up. Microscopic examination of the tissue will settle any doubt as to the nature of the stricture.

By extension and ulceration the growth tends to invade neighbouring organs. Thus, it may so constrict the trachea or bronchi as to lead to suffocation ; it may grow by continuity into the lobes of the thyroid body, into the connective tissue of the mediastinum, or into the bodies of the vertebrae. Ulceration may put the oesophagus into communication with the trachea or bronchi, and give rise to fatal bronchopneumonia ; it may perforate into the pleura or lung—more often on the right side,—and set up pleurisy or gangrenous pneumonia. A gangrenous cavity in the lung was present in 15 out of 85 cases examined at St. George's Hospital. The pericardium is very seldom put into communication with the oesophagus. Thus, in 56 cases of carcinoma of the thoracic portion of the oesophagus, direct invasion of the pericardium did not occur in any instance ; in one case the growth was adherent to the pericardium, and in another there were secondary growths inside the pericardium (Douglas). Ulceration into the aorta or its branches is very rare. Dr. F. Taylor was not able to collect more than 9 examples of this accident. When it occurs, death may result from sudden and profuse haematemesis. The recurrent laryngeal nerves are often implicated in the growth ; and occasionally the sympathetic is similarly affected. In rare cases ulceration of oesophageal carcinoma may be followed by a localised abscess in immediate relation with the growth.

Secondary growths occur most frequently in the adjacent lymphatic glands ; the infiltration may extend against the direction of the lymph-stream. Carcinomatous glands may produce pressure symptoms, such as abductor paralysis and stenosis or occlusion of a bronchus, and even narrowing of the oesophagus when the primary growth is latent. The lymphatic glands close to the fundus of the stomach may be invaded, and I have seen a case in which a mass of carcinomatous glands broke down and, by leaking into the abdominal cavity, set up peritonitis. Secondary growths occur in the lungs and pleurae ; in some instances the bronchus of the affected lung is perforated by the growth, and the secondary nodules may be due to implantation. Metastatic growths may also be met with in the liver, especially in the left lobe, and occasionally in the stomach and intestines, peritoneum, adrenals, kidneys, bones. It is comparatively rare, however, to see secondary growths, except in the immediate neighbourhood of the oesophagus.

Histologically, squamous-celled carcinoma or epithelioma is found in an overwhelming majority of the cases ; spheroidal-celled carcinoma is described in a small percentage of cases. Thus, it was present in four out of fifty-nine cases tabulated by Mr. Butlin. Sir Cooper Perry and Dr. Shaw conclude that primary carcinoma of the oesophagus is always a squamous-celled carcinoma, and that spheroidal-celled carcinoma of the

lower end of the oesophagus is really an extension of a primary growth of the cardiac orifice of the stomach. I have, however, seen primary spheroidal-celled carcinoma of the oesophagus at a distance from the cardiac orifice in cases in which there was no trace of gastric carcinoma. Sir Cooper Perry and Dr. Shaw found that in twenty-four cases of carcinoma of the cardiac orifice the oesophagus was invaded in sixteen, and contested Fagge's statement that carcinoma of the cardiac orifice is really primary in the oesophagus. Very exceptionally colloid carcinoma has been recorded; six examples of columnar-celled carcinoma have been collected by Hewlett. Spheroidal-celled carcinoma may be derived from the mucous glands of the oesophagus.

Symptoms.—These in malignant stricture resemble, in a general way, those of traumatic stricture. Difficulty in swallowing, first of solids and later of liquids, usually comes on gradually; but it may be quite sudden in its appearance. There may be constant dull pain in the gullet, made worse by any attempt to swallow; or severe pain may be felt between the shoulders. But, on the other hand, the dysphagia is often painless, especially when the growth is in the lower part of the oesophagus (Newman). Return of food shortly after it has been taken is frequent; the vomit may contain frothy mucus stained with blood, or even sloughing portions of the growth. Profuse haemorrhage is not common; it may be due to ulceration into the aorta or to erosion of small vessels. In 214 cases of oesophageal cancer tabulated by Hampeln, profuse haemorrhage occurred in twelve, in two of these the source was the descending aorta, in the remaining ten it was assumed to be capillary. Occasionally swallowing becomes easier after separation of projections of the growth which have blocked up the lumen of the tube. On the other hand, the obstruction may suddenly become absolute from impaction of food in the stricture. As a rule the dysphagia is steadily progressive. Cough is a common consequence; and when there is a communication with the trachea or bronchi it is very severe and is constantly set up by taking food. Some cases of primary carcinoma of the oesophagus run their course without any dysphagia, the symptoms being those of the secondary growths. Thus, in two cases at St. George's Hospital, the clinical aspect was that of cancer of the liver, which weighed 14 and 15 lbs. after death; in another case there were widespread metastases in the skeleton.

Hoarseness, enfeeblement, and even loss of voice may result from pressure on the recurrent laryngeal nerves, and are an indication of malignant rather than of cicatricial stricture. Pressure on the cervical sympathetic may produce contraction of the pupil on the affected side. The patients appear anaemic, but from concentration of the blood the number of red blood-corpuscles is but slightly diminished; leucocytosis was present in 5 out of 6 cases examined by Emerson. Hunger in the early stages may be a pressing symptom; but, as the disease progresses, the patient becomes indifferent or even unwilling to take nourishment. There is much discomfort from welling up of fluid into the mouth and

from the presence of ropy mucus in the throat and mouth. The breath becomes offensive. Hiccup may be present, and thirst associated with dryness of the mouth may be a troublesome and severe feature. Rapid emaciation, marked loss of weight, and extreme debility are prominent symptoms.

The progress of the disease is rapid; and death from exhaustion, septic absorption, starvation, or from the various complications, mainly pulmonary, described under Morbid Anatomy, occurs within a year from the first symptoms, often much sooner.

Diagnosis.—The history and rapid progress of the disease will usually distinguish it from traumatic or from simple fibrous stricture; though in the earlier stages, especially if there be no history of the taking of poison, cicatricial stricture may very closely resemble it. Paralysis of one or both vocal cords, or the presence of palpably enlarged glands in the neck, points strongly to malignant stricture.

From chronic oesophagitis, spasmodic stricture, and paralysis, the special features of each and the cautious passage of a bougie will distinguish it. In chronic oesophagitis and paralysis the bougie will pass without any difficulty; but in spasmodic stricture the bougie will be arrested in different situations, and the obstruction can be overcome by persisting in the pressure.

Pain on swallowing, due to inflammation or ulceration about the larynx, may suggest malignant stricture, especially in tuberculous laryngitis when at the same time there is considerable wasting. The passage of a bougie, however, will prove that there is no organic obstruction. In these cases of tuberculous laryngitis liquid food sets up coughing from the entrance of fluid into the larynx, while pulpy or more solid food is taken with less difficulty; in uncomplicated malignant stricture of oesophagus, on the contrary, liquid food is taken more easily than solid, but when carcinoma of the oesophagus has produced a broncho-oesophageal fistula, fluids set up cough. Examination of the larynx will settle the diagnosis.

Occasionally the question as to which of the three conditions—mediastinal growth, aneurysm, or carcinoma gulae—is present, requires most careful consideration of the physical signs and symptoms; and it may be very difficult to come to a definite diagnosis.

Prognosis.—Unless radically removed, and this is practically out of the question, the growth will rapidly cause death. Sir F. Treves' case of survival for nearly three years after gastrostomy for undoubted carcinoma is an example of the vagaries of malignant disease. The course of the disease varies under different conditions, and life may be prolonged by careful feeding through a Symonds' tube, or by an early gastrostomy. When carcinoma attacks the upper part of the oesophagus its course is more rapid than when the lower part is affected. When there is evidence of the extension of ulceration into any of the neighbouring parts, death is at hand.

Treatment.—In a few cases in which the growth was in the neck the

affected segment has been excised; but in the majority of cases it is out of reach, and the results are bad. Opening the oesophagus below the stricture (oesophagostomy) is rarely practicable, is much more difficult, and is less certain than gastrostomy. Practically the treatment is palliative only, and consists in careful feeding and the constant wearing of a Symonds' tube, or in gastrostomy. A free use of alkaline powders affords great relief for the ropy mucus.

In the earlier stages of malignant stricture Mr. Symonds advocates intubation of the stricture. A short tube, with a projecting rim at the top, which rests on the upper margin of the stricture, is introduced through the stricture and left in place: threads attached to the tube are brought out at the mouth, so that the tube can be removed when desired. In the later stages, when there may be a fistulous communication between the oesophagus and the air-passages, it is important, in order to avoid any lung complications, that no food at all should pass into the oesophagus; and this is effected by the patient constantly wearing a tube which is long enough to pass through the stricture into the stomach and to convey liquid food. The upper end of the tube, which is free at the back of the mouth, is fixed by threads to the whiskers, to the skin behind the ears, or to the teeth. The condition of the threads should be examined daily, and care taken that they are not bitten through.

Gastrostomy is usually resorted to much too late in the course of the disease, and thus the operation has not a fair chance. This is well borne out by Mr. Newman's statistics of gastrostomy both for cicatricial and malignant stricture. Of 48 cases of cicatricial stricture, 23 died within a month of the operation; and of 280 cases of gastrostomy for malignant disease, 154 died within a month. The causes of death in both classes taken together of those dying within the month were—exhaustion in 53 per cent, peritonitis in 21 per cent, pneumonia in 15 per cent.

On the whole, gastrostomy is the most satisfactory method of treatment, and should not be delayed until the patient is emaciated and pulled down by chronic starvation. Yet at best gastrostomy is but palliative, inasmuch as the growth gradually invades the important structures in immediate relation with it, and thus leads to death in one or other of the various ways already mentioned. It is therefore hardly fair to compare the effects of gastrostomy with those of colotomy for carcinoma, and to expect that life will be as much prolonged in the former case. Einhorn and Exner have employed radium, introduced into the oesophagus, with some relief to the symptoms. As might be anticipated, *x*-rays applications to the surface of the body have not benefited a malignant stricture of the oesophagus (Einhorn).

Innocent Tumours.—Simple or benign tumours of the oesophagus are rare if the small warty growths, which are quite unimportant, be excepted. Fibromas, fibrolipomas, and myomas may be of considerable size, and may simulate the symptoms of cicatricial or malignant stricture; on the other hand, from compensatory dilatation of the oesophagus

around the tumour (which is almost always polypoid) there may be no obstruction; and symptoms may be absent.

Other growths, such as lipomas and peri-oesophageal cysts, have been described, but are mere pathological curiosities.

Peri-oesophageal Abscess.—As the result of caries of the spine an abscess may be formed in connexion with the oesophagus after the manner of a post-pharyngeal abscess; but a peri-oesophageal abscess frequently presents laterally, and displaces the oesophagus to one side and not anteriorly. Disease of the gullet, such as ulcer, rupture, or carcinoma, may lead to peri-oesophageal abscess.

Suppurating glands, whether secondary to inflammation elsewhere, as in the mouth, throat, or head, or as the result of tuberculosis, may lead to an abscess in close relation to the oesophagus. A caseous gland at the bifurcation of the trachea may soften down and discharge into the oesophagus, or into the trachea; or may lead to a fistulous communication between the two. Cicatrisation of the abscess cavity left after discharge into the oesophagus may result in a traction diverticulum. Suppuration and abscesses, from whatever cause they arise, may open into the oesophagus; thus, an abscess in connexion with the larynx or thyroid gland may discharge into it. Suppuration in the immediate neighbourhood of the oesophagus may follow acute traumatic oesophagitis, or may result from ulceration of the oesophagus, due to impaction of foreign bodies.

The abscess may be of very various dimensions; eventually it discharges into the oesophagus: while eroding the oesophageal wall it may travel in the loose submucous layer, and dissect the mucous coat from the muscular coat, and thus give rise to a diffuse phlegmonous oesophagitis. Except when due to disease of bone the course of a peri-oesophageal abscess is acute.

Symptoms.—The chief symptoms are pain in swallowing, with difficulty in taking food, and pain on movement of the neck. Dyspnoea, due to pressure on the trachea, may come on.

Treatment.—If the abscess is in the neck it should be opened at once. A peri-oesophageal abscess in the thorax in connexion with the bronchial glands cannot be diagnosed with any certainty; but it has been suggested that emetics should be given in the hope of rupturing the abscess by the effort of vomiting.

Varicose Veins.—In portal obstruction, as in cirrhosis, all communications between the radicals of the portal vein and the general systemic venous system become dilated. There is an anastomosis between the gastric and oesophageal veins, whereby the blood can pass from the stomach into the azygos veins without having to traverse the liver. Varicosity of the veins at the lower end of the oesophagus may, however, occur without any morbid condition of the liver. It is of course rare, but cases have been recorded in which fatal haemorrhage in quite young patients was due to this cause. These veins may become considerably dilated and varicose. Rupture or ulceration of these oesophageal piles has led to fatal haemorrhage; usually the blood passes into the stomach

and may be vomited up: in some rare instances the blood flows out of the mouth and is not ejected as in haematemesis.

Treatment of the haemorrhage should consist, like that of ulceration of the oesophagus, in rest to the part, at first no food should be given by the mouth, and subsequently in very careful feeding and avoidance of any rough or irritating food.

Neuroses of the Oesophagus.—*Spasmodic Stricture of the Oesophagus.*—*Oesophagismus* occurs in neurotic young women (18-30 years); but also in men of a similar type, and in hypochondriacs. Cases have been recorded in which it has attacked mother and daughter in such a way as to appear hereditary.

It may be purely psychical; but it is more often, perhaps, associated with reflex irritation, especially in the area supplied by the vagi, if not secondary to it. Thus, as might be expected, spasm may accompany any disease of the oesophagus itself; especially inflammation and ulceration. But in these cases, inasmuch as there is some organic change in the gullet, the condition of spasm is secondary or subordinate, and is not to be described simply as spasmodic stricture. Characteristic examples of this neurosis may seem to depend on pharyngitis. It may depend on gastric disorder of no great severity, but it may be associated with carcinoma, and is sometimes dated back by the patient to damage received in swallowing rough food, bones, or foreign bodies; it is then perhaps due in part to "auto-suggestion." Reflex irritation from more distant parts, such as a floating kidney or the genital organs, appears to play some part in setting up spasm. Spasm of the oesophagus has been associated with the abnormal origin of the right subclavian artery from the thoracic aorta. This vessel, which is due to persistence of the right 4th aortic arch, passes behind, sometimes in front of the oesophagus, and has been thought to compress that tube and thus to give rise to "dysphagia lusoria." Whether this be so appears doubtful. Spasm is a prominent and painful feature in human rabies, and is present in the hysterical imitation of that disease. It has been attributed to the gouty and rheumatic diatheses.

Pathology.—Spasm may occur in any part of the oesophagus and may be transient or persistent. An irregular contraction of the circular coat has been supposed to precede the bolus of food instead of accompanying or following it, as normally it should do. In persistent spasm of the sphincter fibres at the lower end of the oesophagus, or cardiospasm, food is retained, and as a result dilatation of the gullet follows (*vide* p. 333).

Symptoms.—In the slighter forms the dysphagia, which is more or less paroxysmal and varying in degree, comes on suddenly, and may be attended by gulping noises. There is no inclination for food, and when taken it may be suddenly and violently rejected. Spasm is increased by cold liquid food, so that the patients usually prefer warmed food. Globus hystericus may be present, and be accompanied by some pain and discomfort. As emaciation is not present, or advances to no great degree, its absence helps us to distinguish this affection from malignant stricture. There is no constancy in the spot where the bougie is arrested; the

resistance will nearly always disappear as the pressure is continued, if necessary under the influence of chloroform. The symptoms and treatment of persistent cardiospasm have already been described, p. 335.

Diagnosis.—The age, sex, and character of the patient should be taken into account. The manner in which the dysphagia comes on, and its intermittent or progressive course, are important in distinguishing spasmodic from organic spasm. The absence of emaciation is strongly in favour of spasm. In addition to the knowledge to be obtained from the passage of a bougie, to which I have already referred, auscultation of the oesophagus may be employed. In spasmodic stricture the oesophageal sound is unaltered; in organic stenosis it is prolonged, delayed, or replaced by a continuous confused bubbling sound, sometimes with a grating note.

Treatment is local and constitutional. The passage of a bougie may bring about a cure, or cautious galvanism by means of an oesophageal bougie may be successfully employed. The local application of cocaine has been recommended.

Liquids, not solid food, should be given; and care should be taken to see that they are warmed and sweetened. General treatment of the patient's health and special anti-hysterical and antispasmodic remedies should be prescribed, and attention directed to the cure of hysteria or hypochondriasis.

Paralysis of the oesophagus is a decidedly rare affection.

Etiology.—It may be functional or hysterical in origin; but in hysteria spasm is much commoner than paralysis. It may occur in cerebral tumour and general paralysis of the insane.

Diseases attacking the nuclei in the pons and medulla, as in bulbar paralysis, lesions of the vagi, and peripheral neuritis due to lead or the poison of diphtheria, may occasionally produce it. It has been attributed to muscular weakness, and difficulty of swallowing occurring in the course of acute fevers may thus be explained.

Morbid Anatomy.—Little is known of the morbid anatomy of the affection: Morell-Mackenzie believed that the potential lumen of the tube becomes diminished and the walls degenerated. On the analogy of other paralysed viscera dilatation would have been expected. Possibly some cases of simple dilatation of the oesophagus, in which there is no apparent cause, such as a stricture, may be thus explained.

The symptoms consist in a difficulty in swallowing food, which, however, though constant, is not absolute unless the pharynx be affected. The normal oesophageal sound, as heard by auscultation, is altered or lost. Passing a bougie proves the absence of any obstruction or stricture. Regurgitation of food, which is common and copious in dilatation, seldom occurs in paralysis.

The *treatment* should, when possible, be applied to the primary cause; but local stimulation by pungent food, the passage of a tube, or electricity, may be tried. Faradisation of the interior of the oesophagus must be carried out with caution, as the vagi lie in close proximity. It

is obviously more likely to succeed in the hysterical cases than in those the result of grave organic diseases of the nervous system.

In cerebral tumour or bulbar paralysis care must be taken to see that a sufficiency of food is taken, and, if need be, the patient should be fed by the stomach-tube.

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DYSPEPSIA

By Sir Lauder Brunton, M.D., F.R.S.

Dyspepsia in Childhood

By W. Soltau Fenwick, M.D.

Dyspepsia is a general name which is generally used as synonymous with indigestion. It properly means discomfort or pain during digestion in the stomach; but it is frequently used to signify discomfort or pain during the process of digestion, whether in the stomach or intestines. The vagueness of its significance has both advantages and disadvantages. The disadvantages of the name are that it signifies so many different conditions that it is little or no guide to the exact functional or organic lesions of the several organs concerned in the digestive process; and without a more or less accurate knowledge of the nature of a disease we cannot hope for success in treatment. Its advantages, on the other hand, are that it includes a group of symptoms which cannot always be referred to an exact physiological or anatomical basis. In this respect it corresponds to albuminuria or jaundice, affections which, although they can often be assigned correctly to their causes, yet sometimes defy exact diagnosis. It may also be compared with the symptom of itching in the skin, which may be due to eczema, urticaria, pruritus; or, as in jaundice or Bright's disease, to the presence of abnormal constituents in the blood. If we had to depend for our diagnosis solely on the patient's account of his feelings, we could not distinguish these affections; nor could we do so successfully even if we were able to palpate the surface. By ocular inspection we may frequently distinguish them; but if, instead of being exposed to view, the skin were inside the body, it is evident that the diagnosis would often be impossible. Thus it is with many cases of dyspepsia. The patient complains of great discomfort, but we are unable to discover any organic cause; and frequently are not able to decide even upon the nature of the functional derangement on which the symptoms depend. In some cases the derangement is probably very slight, although the symptoms may be severe; just as itching in eczema, although the irritation is confined to the surface of the skin, may render the patient's life almost intolerable. Some cases of dyspepsia very probably depend upon a similar kind of irritation of the gastric or intestinal mucous membrane; for occasionally eczema will disappear and indigestion take its place, and conversely.

As medical knowledge advances the use of the name dyspepsia will be greatly restricted (1), and we shall be able to assign the different forms of indigestion more and more exactly to their proper causes. At present, however, it must be retained.

Foods.—In order to understand the symptoms of dyspepsia we must first consider the various classes of foods, and the mode in which they are

normally digested. In place of simply tabulating the various classes of foods, it is easier to suppose an ordinary mixed meal, consisting of beefsteak with fat attached, bread or potato, salt, pepper, mustard, pickles, fruit stewed with sugar, and alcohol in beer, wine, or spirits. The lean part of the beefsteak is a good example of the protein constituents of food; the fat of the fatty constituents; the bread or potato of the carbohydrates; the salt of the mineral constituents, of which others are contained in the meat itself and in bread. Water is the largest and one of the most essential ingredients of the body. The stewed fruit and vegetables contain cellulose and neutral salts of vegetable acids combined with potash or lime. The sugar, like the bread and potato, belongs to the class of carbohydrates; tne pickles contain organic acids with cellulose; and mustard and pepper contain pungent oils, which have the power of irritating, with more or less energy, any mucous surface with which they are brought in contact. Alcohol has a similar power of local irritation, which, however, is modified very greatly by its dilution; moreover, after its absorption, it has a marked influence on the nervous system and circulation.

Symptoms.—In health we are unconscious of the existence of any part of our body save in so far as we get pleasure from its exercise; and this is true of the digestive organs as well as of others. For in perfect health the only sensations connected with digestion are those relating to the ingestion of food and the ejection of excreta; and the slight inconvenience they occasion is more than compensated by a sense of relief in the latter case, and the positive pleasure associated with the former. In some cases of dyspepsia the symptoms are associated more with the palate than with the stomach; in other cases rather with the intestines. The pleasure afforded by eating is frequently referred to the palate; although it probably depends to a great extent upon the state of the stomach. In dyspepsia the normal desire for food occasionally gives place to a ravenous craving; but more frequently it leads to a loss of appetite, distaste for food, nausea, or actual vomiting. The presence of food in the stomach, instead of being unfelt as in health, gives rise to a sense of oppression in the epigastrium, disagreeable eructations of gas, food, or acid, and feelings of weight, oppression, uneasiness, discomfort, distension, or pain. The pain may be diffused; it is very often situated at the junction of the oesophagus with the stomach opposite the end of the sternum, when it is known as heartburn. In the intestine, likewise, there may be uneasiness, pain, distension, passage of flatus, and either diarrhoea or constipation. In addition to these symptoms there is sometimes a great flow of saliva from the mouth, or the ejection of a large quantity of watery fluid, which is mostly saliva although it appears to come from the stomach. This is frequently accompanied by very acid eructations, the acid being such as to give rise to a burning in the mouth; and from the burning in the epigastrium and mouth, with which the flow of watery fluid is frequently associated, the symptom has received the name of pyrosis, a name which is now given to this ejection of water even although no feeling of burning be present.

Briefly, the symptoms of dyspepsia are a furred tongue, a bad taste in the mouth, want of appetite or even loathing of food, nausea, vomiting, oppression in the chest, weight at the epigastrium, pain, distension, flatulence, acidity, eructations, pyrosis, constipation or diarrhoea. The flatulence either passes upwards in belching or downwards per anum, or, if it remain in the stomach and intestines, it gives rise to distension.

Analysis of Symptoms.—*Furred Tongue.*—The tongue is usually taken as a useful index to the condition of the mucous membrane of the alimentary canal. In health it ought to be clean; but in disease it is frequently covered with a more or less thick coating of a white, yellow, brown, or even black colour. This coating consists chiefly of epithelium with masses of micro-organisms. Its thickness and colour depend to a considerable extent upon the diet, and if the diet be very soft—especially if it be liquid, as in the case of the milk diet usually employed in enteric fever—the tongue has a thick, white, creamy coating. When the diet is solid the food tends to rub off the accumulations of epithelium, and to keep the tongue clean. But the growth of microbes on the tongue is greatly stimulated by decomposing food; and this may be well seen in the case of persons with a decayed molar tooth, in which particles of food accumulate; the decomposing food in the hollow tooth seems to act as a kind of manure for the microbes on the tongue, and these consequently grow luxuriantly, giving rise to a thick patch of fur on the tongue close to the decayed tooth; even though the rest of the tongue may be clean. How far the thick fur which occurs in some cases of dyspepsia, or in fevers, is due to alteration in the secretions of the mouth, and how far to trophic changes in the mucous membrane of the tongue itself, is not certainly known. Usually, however, a white pasty fur on the tongue is looked upon as a sign of atony and weakness in the digestive tract; while a pointed tongue with large papillae, clean and rather red at the edges and tip, is regarded as a sign of irritation and an indication for soothing treatment; a pale, flabby tongue is supposed to indicate the necessity for stimulating and tonic treatment. A bad taste in the mouth is sometimes due to decaying teeth or to artificial teeth or plates that are worn too constantly, and are not sufficiently purified by antiseptics; but in addition to this, we know that a bad taste may arise from bitter substances given as medicine. Not only does quinine produce a bitter taste in the mouth when swallowed, but if administered with iodide of potassium the bitter taste may be persistent during the whole day. The iodides alone give rise to a saline, disagreeable taste, of which many patients complain; this is caused by absorption from the stomach and re-excretion into the mouth through the salivary glands. When quinine is given at the same time the iodide appears to carry the quinine along with it into the mouth, and thus to give rise to the bitter taste. Bitter substances are formed in the body naturally, even when no medicines are taken; and although fresh healthy bile is tasteless, bile is sometimes excessively bitter. The cause of the bitterness has not been exactly ascertained; but it is probably due to accumulation of those bitter substances formed during

digestion, which frequently make milk disagreeable to the palate when artificially peptonised for too long a time. It is probable that these bitter substances reach the mouth in the same way as iodide of potassium or quinine.

Disagreeable Breath.—A disagreeable smell of the breath, like a furred tongue or unpleasant taste, may be due to decomposition of food in carious teeth; but in some cases it is almost certainly due to the elimination of substances which have been absorbed from the intestine and are excreted through the lungs. Sulphuretted hydrogen, when injected into the intestine, is absorbed with great rapidity and eliminated through the lungs; and the same is the case with such volatile vegetable oils as those of onion and garlic.

Appetite, although closely associated with the gratification of the gustatory nerves in the mouth, is yet more intimately dependent upon the condition of the stomach, and upon the wants of the body as a whole. The exact cause of it has not yet been ascertained, but certainly it is closely connected with the circulation of the blood in the stomach. The mucous membrane of this organ during fasting was observed by Beaumont to be pale; but on the ingestion of food it became rosy at once, and secreted gastric juice. It is probable that appetite consists in a condition of the nerves of the stomach similar to that which is produced in the nerves of the skin by itching or gentle tickling; and is not improbably connected, to some extent, with distension of the lymph-spaces in the mucous membrane, just as in the skin a sensation of itching frequently precedes the outbreak of sweat. When the stomach is somewhat weak or atonic there is frequently no desire for food at first; but after a little has been taken the appetite comes with the eating until a fair quantity has been consumed. Sometimes Beaumont observed that a craving appetite was associated with a red and irritable mucous membrane on which even some abrasion of the surface was present. When, however, irritation of the stomach, mechanical or chemical, is carried too far, the normal secretion of gastric juice stops, the circulation becomes lessened, the mucous membrane turns white, and mucus is secreted. Still further irritation causes retching and vomiting.

It is evident that, if appetite be thus closely associated with the condition of the circulation in the stomach itself, the appetite and the circulation together are likely to be influenced by the state of the liver; for nearly all the blood from the stomach must pass through the liver before it can return into the general circulation. If this organ be congested, and the circulation through it slow, the appetite is likely to fall into abeyance. It must be borne in mind also that various substances have the power of stimulating the walls of the stomach in such a way as to produce increased appetite, anorexia, or vomiting. Many substances which in small doses increase the appetite are emetics when given in large doses. Thus, a little mustard or horse-radish, a little salt or other condiment taken with food may render it more palatable; a bitter tonic taken before a meal will increase the appetite; but all these sub-

stances taken in large quantities act as emetics. A very minute quantity of arsenic is a powerful stimulant to the appetite, but a large quantity produces most violent vomiting.

In dyspepsia, then, we may have a craving appetite, a want of appetite, or an appetite for abnormal kinds of food. The craving appetite, according to Beaumont's experiments, appears to go with an irritable condition of the stomach, such as might be caused by small doses of arsenic. It is very frequently noticeable as the first stage of what is called "a bilious attack"; and when the irritation in the stomach becomes greater, the craving appetite gives place to anorexia, and is followed by nausea, or even by vomiting. Lack of appetite may be associated with two conditions: either with the absence of tone and deficient circulation through the stomach, or with excessive irritation bordering upon nausea. In the first case, where lack of appetite is dependent upon weakness and absence of circulation, the person has little or no desire to take food; but after beginning to eat, the gastric circulation becomes brisker, appetite comes with eating, and the person may be able to take a fair meal. In the case of a craving appetite, however, things are different; and the stimulation by food, which in the case just mentioned brings on the appetite, stimulates the irritable stomach to excess, so that appetite very quickly disappears; if, then, more food be forced down, nausea or vomiting ensues. Where the stomach is already so irritable that nausea is felt before the food is taken, vomiting is very likely to come on immediately after eating. A lack of appetite restored by eating, therefore, indicates an atony of the stomach, which is likely to be benefited by tonics and stimulants; a craving appetite easily satisfied, or anorexia with a tendency to nausea in eating, indicates irritation and requires sedative treatment. It must be remembered that various poisons injected into the veins are excreted by the walls of the stomach; thus, tartar emetic, morphine, and serpent's venom injected into the veins are all excreted by the gastric mucous membrane; and the same is the case with poisons formed in the body, such as the toxalbumins of cholera, and possibly also toxalbumins in cases of renal disease. It is most important to bear in mind that disturbance of the appetite may be due to poisons formed in the intestine or in the tissues and excreted into the stomach, where they give rise to disturbance; as we observed that they do in the mouth. The elimination of these poisons from the body is, therefore, to be carefully attended to as a means of restoring appetite.

Vomiting is the expulsion of the contents of the stomach through the oesophagus and mouth. It is effected by the squeezing of the stomach between the diaphragm and the abdominal walls. This squeezing is ineffectual to empty the stomach if the cardiac sphincter be contracted; when the movements are simply known under the title of "retching." When the cardiac orifice dilates, and simultaneously the diaphragm descends to its utmost, and the abdominal muscles contract, the contents of the stomach find an issue through the oesophagus, and vomiting occurs. The nerve-centre by which the movements of the stomach, diaphragm,

and abdominal muscles are so co-ordinated as to produce vomiting is situated in the medulla oblongata, and is closely associated with the respiratory centre, some nerve-cells being in all probability common to both centres. The vomiting centre may be stimulated sometimes by poisons, such as tartar emetic or apomorphine which act upon it directly; but it is usually brought into action reflexly by irritation which may start from various parts of the body, and is conveyed to it by the afferent nerves. When stimulated from the brain, as in violent emotion or in organic disease—such as tuberculous meningitis or cerebral tumour—the vomiting is sudden and not associated with nausea; and the same is the case when it is stimulated from the pharynx by the tickling of a feather. Even stimulation from the stomach may be associated with very little nausea; mustard and hot water cause vomiting speedily, but with very little antecedent nausea; tartar emetic, on the other hand, causes much nausea. Vomiting is associated also with irritation in the liver, kidney, intestines, and genital organs; so that during the passage of a gall-stone or of a renal calculus there may be violent vomiting; and when vomiting is persistent the presence of a hernia should always be looked for. The vomiting of pregnancy is usually supposed to be reflex from the uterus, but it may be due to the presence of toxalbumins, as in cholera. Vomiting in dyspepsia is usually due either to the condition of the contents of the stomach or to that of the gastric walls. Thus, it may occur after too bulky a meal, when the food, although perfectly wholesome, has been taken in such large quantity that the stomach is unable to digest it. Or it may occur from the ingestion of food which is either irritating in itself, or is apt to undergo such decomposition as to lead to the formation of irritating products.

Thus, tainted meat or fish, an oyster of doubtful quality, or bad mushrooms may cause vomiting very shortly after their introduction; while a mixture of *pâté de foie gras* with milk and beer may yield irritant products, and give rise to vomiting some hours later; although each article was good in itself, and the quantity taken not excessive. The food which has been taken into the stomach is much more liable to undergo decomposition with formation of irritating products if delayed in the stomach. This is especially noticeable in cases of contracted pylorus, whether in consequence of malignant disease or from cicatricial contraction due to old ulcers. In such cases the food remains many hours in the dilated stomach, and is then vomited; usually in a state of fermentation. But even in health imperfectly masticated food may remain too long in the stomach, as the lumps in it are not readily dissolved by the gastric juice. When these lumps attempt to pass the pylorus they stimulate it to contraction, and thus not only is their own passage arrested, but a quantity of other food also is kept back with them. Thus, pieces of potato, of apple, of cheese, or even of butcher's meat, which have been swallowed without being comminuted by the teeth, are again and again forced to the pylorus by contractions of the stomach; but not being able to get through, they are ultimately rejected by vomiting. In cases of sick headache, a transient

condition of dilatation of the stomach, and probably of contraction of the pylorus, occurs—the condition being, in fact, one of a proximal dilatation with peripheral contraction, similar to that which I have pointed out in the arteries of the head during migraine.

The condition of the gastric walls is a most important factor in vomiting. When the mucous membrane of the stomach is rubbed too hard with a piece of glass rod, the natural rose colour disappears, the secretion of gastric juice stops, mucus is secreted, and vomiting is set up. Here the vomiting is, no doubt, simply reflex, as in the case of vomiting by tickling the fauces with a feather; but in Beaumont's observations on Alexis St. Martin we notice that great irritability of the mucous membrane of the stomach tended to give rise to nausea and vomiting on taking food. In some cases, no doubt, there is hyperaesthesia of the mucous membrane of the stomach, just as there may be of the fauces. The condition of the stomach in sick headache has already been noticed. A common cause of vomiting in the morning is the practice of taking an excessive quantity of spirits at night before going to bed. Postnasal and pharyngeal catarrh is not uncommonly a cause of retching or vomiting on rising in the morning. This may occur in persons who are most abstemious in the use of alcohol, and who may not have touched it in any form for weeks or months. When the nasal passages are cleared of mucus the retching usually ceases.

Vomiting usually occurs during or immediately after a meal, when it is of purely nervous origin, during the height of digestion when due to gastric ulcer, and after some hours when there is dilatation of the stomach.

Oppression.—The feeling of oppression, which is generally referred to the cardiac region, is in most cases due to distension of the stomach with flatulence; but in some it is no doubt due to reflex irritation of the vagus acting upon the heart; indeed, the pulse may be much slowed by gastric irritation.

Pain in the stomach varies very much both in extent and character. In simple dyspepsia, not associated with ulceration or malignant disease, it is usually either of a dull character, extending over the whole epigastrium, or is a burning sensation nearly opposite the end of the sternum. The burning is closely associated with great acidity of the contents of the stomach, so that, when these regurgitate into the mouth, they seem to give rise to a burning feeling in the throat and set the teeth on edge. The point at which this burning is felt is just at the junction of the oesophagus with the stomach; and I am inclined to think that it is due to irritation not of the stomach itself but of the lower end of the oesophagus by the acid contents of the stomach.

Acidity.—I have observed that when there is much acid in the stomach this pain may sometimes be brought on or relieved at will, simply by turning from one side to the other; on turning to the left side, so that the gastric contents fill the fundus and do not reach the cardiac orifice, the pain may disappear; on turning to the right side, so that the

contents of the stomach fill the smaller pyloric end, and are likely thus to reach the cardiac orifice, the burning sensation may at once reappear. I have noticed, too, that the burning sensation may come on almost immediately after drinking a cup of tea, although this liquid is neutral in itself and not irritating to the oesophagus. The tea may act simply by increasing the volume of the contents of the stomach, so as to make them reach the cardiac orifice, and irritate it by their acidity; but it seems very improbable that during the natural movements of the stomach the gastric contents should not pass over the cardiac orifice; it seems to me more probable that tea causes a protrusion of the mucous membrane of the oesophagus into the stomach, much as the mucous membrane of the rectum occasionally protrudes through the anus after the administration of an enema; the mucous membrane of the oesophagus, which is much more sensitive than the mucous membrane of the stomach itself, would thus be exposed to irritation by the acid contents of the stomach, and would feel the burning. When we swallow anything hot, the burning which it occasions ceases just at the point where the hot liquid or solid leaves the oesophagus and passes into the stomach. This acidity of the gastric contents is not due, I think, in the majority of cases to any excess of hydrochloric acid, but rather to organic acids, such as lactic and sometimes butyric acid. Butyric acid seems to have an especially irritating local action. Although lactic acid appears to be a normal constituent of the gastric juice, yet excess either of it or of any other organic acid is generally due to fermentation of sugars, alcohols, or fats.

There are, however, numerous cases in which a gastric juice containing an abnormally high proportion, twice or even three times the normal, is actually secreted. To this condition the name of *hyperchlorhydria* has been given. The acidity of the gastric juice varies in different individuals and at different periods of digestion, and in some persons a degree of acidity appears to be compatible with perfect health which in others gives rise to acute discomfort. There appear, therefore, to be two factors in it, (*a*) increased proportion of hydrochloric acid, and (*b*) increased sensitiveness of the stomach to it. Both of these conditions appear to be associated with an abnormal sensitiveness of the nervous system, and the condition may, therefore, be regarded as a typical form of nervous dyspepsia. Excessive acidity may occur in apparently healthy persons from irritating articles of food which may not be in themselves objectionable, but which may be hastily swallowed, imperfectly masticated, they may be too hot or too cold, or they may be taken in too great quantity, especially certain things, *e.g.* strong coffee, alcohol, or condiments. But in the condition of hyperchlorhydria the secretion of acid is more readily induced, and the stomach appears at the same time to feel it more. It occurs chiefly in patients of a nervous temperament, and is very frequent in chlorosis. It may come on suddenly from emotional disturbance (gastroxynsis of Rossbach). It causes oppression in the epigastrium, coming on an hour or more after eating, and this may

increase till it amounts to very acute pain, which is relieved by the stomach emptying itself either into the duodenum or by vomiting, or by the neutralisation of the acid by more food, especially protein foods, *e.g.* flesh, eggs, or milk, or by alkalis. The extra acidity of the gastric juice interferes with the digestion of amylaceous food, but hastens the digestion of proteins, and the appetite is, as a rule, good, and patients often wish to eat at short intervals. The condition may be intermittent, coming on for a few days, and then disappearing, or it may be continuous, and it is then difficult or impossible to diagnose it from gastric ulcer.

Closely associated with this is the condition in which the secretion from the stomach is excessive in quantity, and may occur in an empty stomach. To this the name of *gastrosuccorrhoea* has been given. In many cases not only is the quantity of gastric juice increased but the proportion of hydrochloric acid, as in the condition just described. It may occur in an intermittent or continuous form. In the intermittent form it resembles hyperchlorhydria, but differs from it chiefly in the fact that vomiting is more common in gastrosuccorrhoea, and after the vomiting has cleared the stomach of food large quantities of acid fluid, often bile-stained, are ejected. The symptoms of oppression and pain also come on not only after a meal but when the stomach is completely empty, so that pain and vomiting of acid liquid occur in the night. In the continuous form the vomiting is more constant, the stomach frequently becomes dilated, and the patient loses flesh and strength so much that cancer of the stomach may be suspected. In gastrosuccorrhoea the vomited matter usually falls into three layers, a lower one of starch grains, a middle one of turbid liquid, and an upper one of froth, but there are few or no remnants of flesh. The diagnosis can only be made with certainty by washing out the stomach and then finding that the empty stomach secretes excess of gastric juice.

Pyrosis.—Acidity in the stomach gives rise to too frequent desire to swallow saliva, which, by its alkalinity, eases the irritation of the lower end of the oesophagus. Not only so, but a reflex secretion of saliva appears to be induced in the salivary glands by gastric irritation ; so that the saliva either runs from the mouth or is swallowed frequently, and accumulates either in the stomach or in the lower part of the oesophagus, and is then ejected in considerable quantities as a nearly colourless, slightly alkaline, and opalescent fluid, the watery character of which has given rise to the popular name of "water-brash."

Eructations of acid fluid may also occur, and the acidity may be so great as to set the teeth on edge and give rise to a feeling of scalding in the throat.

Flatulence.—Wind in the stomach and intestines may be due either to increased ingestion, increased formation, diminished absorption, or lessened expulsion of gas. A small quantity of air is usually swallowed with the food, and a good deal is swallowed with saliva. Thus, if irritation in the stomach lead to frequent swallowing of saliva, more air is swallowed with it, and either gives rise to distension or to belching.

Along with this wind small quantities of liquid or of food may come up into the mouth.

It is evident that anything that lessens fermentation in the stomach, and thus stops the formation of acid, will also lessen the flatulence which is due to the frequent deglutition of saliva. The air which is swallowed, when it is not again brought up, undergoes partial absorption, the oxygen being quickly absorbed, while the nitrogen remains in the stomach and intestines. There is, however, another source of gas in the decomposition of food, and especially of the carbohydrates, which gives rise to the formation of marsh gas. This gas may be derived from both sugars and starches, but it is yielded in larger quantities by cellulose; so that particular articles of diet containing much cellulose are apt to cause flatulence, and when sulphur also is present—as it is in most of the cruciferae, such as cabbage—sulphuretted hydrogen may also be found.

The occurrence of constipation and diarrhoea from changes in the intestine will be discussed later.

Causation.—The causes of dyspepsia may be divided into (A) those which concern food, and (B) those which concern the organism.

The food may be wrong either in quantity or quality.

Quantity.—If food be habitually taken in too large amounts, or at too frequent intervals, the digestive organs will be unable to cope with it, and indigestion will ensue. The quantity which different persons are capable of consuming varies within wide limits; one man may eat in a day as much as would serve another for a week. Moreover, the quantity that can be consumed by the same individual varies very much in different circumstances, as with the time of the year, exposure to cold or heat, and, more especially, the amount of exercise which can be taken, and the nature of the exercise, whether it be in a confined place or in the open air.

Meal-Times.—The times at which food is taken may also lead to indigestion; not only may the meals be altogether too frequent, but they may be too frequent at one time of the day and too far apart at another. Some patients, for instance, take all the food of the twenty-four hours between 8 A.M. and 4 P.M., and nothing else during the rest of the twenty-four hours; others, again, take their meals with great irregularity, sometimes lunching at 1 o'clock, sometimes fasting from 7 in the morning till 9 at night. Even if there be no such irregularity as this, the time of meals may vary from half an hour to an hour and a half.

Temperature.—The temperature at which food or drink is taken may also be productive of dyspepsia. Thus, iced water, by chilling the stomach and lessening both its secretion and movement, may cause indigestion; and very hot tea may possibly have a similar effect.

Disintegration.—The mechanical disintegration of food has much to do with its ready digestion; and when it either is not or cannot be thoroughly disintegrated it will be slow of digestion. If any one wishes to convince himself of this, let him repeat the test for pepsin in the British Pharmacopoeia, using for comparison in one glass, hard-boiled

egg, in pieces as large as can conveniently be inserted, with pepsin and acid, and in another glass, pieces of the same egg rubbed up into a fine powder. He will then easily understand how slowly large lumps are digested in comparison with the same substance when finely comminuted.

One great cause of imperfect comminution is hurry at meals. The pressure of business frequently compels people to spend as little time as possible over their food, and thus it is bolted rather than swallowed, and is masticated very imperfectly indeed. I have seen a man stick his fork into a new potato, about an inch long and half an inch across, swallow it whole at one gulp, and then complain that new potatoes were indigestible. Another cause of imperfect mastication is decay or absence of the molar teeth. On account of decayed and tender teeth on one side of the mouth the whole of the work may have to be done by the grinders on the other side. If the molars be tender on both sides, mastication must be done by the incisors, and that imperfectly; or perhaps it is not done at all. Not infrequently we find, too, that the molars, if not entirely absent, are deficient in such a way as to have almost no grinding surface. When the two surfaces meet one another over so small an area they do practically nothing more than keep the jaws apart and allow the food to escape between them.

Quality of Food.—Another condition is the quality of the food. Thus, flesh of various kinds may be hard and difficult to masticate; either from the age of the animal, from the meat having been cooked while still in a state of rigor mortis, or from artificial hardening by salting or smoking. If any one will take a piece of beef and tear the fibres apart, he will readily see why it is less digestible than mutton; for while beef tears into cords, mutton tears into fine threads: in the breast of a fowl the threads are still finer. In fish the fibres are not only fine, but shorter than those of flesh or fowl; and in small fish, such as whiting and smelt, the fibres are finer than in such fish as cod. Properly boiled meat or fish is softer and more easily disintegrated than the same meat or fish roasted, broiled, or fried. Although the breast of a fowl is easily broken up, the same is not the case with the leg; and the leg of a tough old fowl may be very much harder to masticate than a slice of roast beef or mutton. Skin is hard to masticate, whether it be the skin of fish, flesh, or fowl; it is hard to digest, therefore, and the same is the case with sinews and ligaments. In small fish, such as whitebait, the skin cannot be removed, nor can the bones; and the presence of the skin and the bone together makes such fish very indigestible.

Fish, flesh, or game that has become high or tainted is indigestible from a different cause; namely, from the presence in it of microbes or of poisons formed by the decomposition of protein substances which may give rise to violent local disturbance of the digestive canal, or to symptoms of general poisoning. Curries, although very palatable, are somewhat dangerous, because the pieces of which they are composed are apt to be eaten without thorough mastication; and occasionally splinters of bone may be swallowed. Some kinds of farinaceous food are by no means easy to

disintegrate; as, for example, soft rolls and buns, and newly baked bread. Any one can see this for himself by contrasting the behaviour of a piece of new bread with the same bread when stale. If new bread be rolled between the finger and thumb it becomes a dense, coherent mass; the stale bread treated in the same way is disintegrated into fine powder. A piece of dry toast is brittle, and breaks readily into powder between the teeth, and its brittleness is not impaired by spreading butter upon it just before eating; but if the same toast, with butter spread upon it, be laid before the fire so that the butter soaks in, it becomes much tougher and less easily broken up: thus to stand in the heat with butter upon it may convert the toast from a readily digestible into an indigestible article of food.

Pastry is usually put down as very indigestible; and the pastry of the pastry-cook is certainly very apt to cause dyspepsia; but pastry carefully made at home may often be eaten with impunity. The reason of this probably is that if the pastry be made with the very best butter, and rolled very fine so as to make it flaky, it is neither injurious mechanically nor chemically; but if it is heavy it will not be readily broken up, and if the abominations sometimes known as "cooking-butter" are used in its composition, it will be very apt to disagree chemically. Many people think that a piece of bad butter, which it would be impossible to eat if it were spread upon a piece of toast, will do no harm if incorporated in pastry, because it is not so easily detected by the sense of taste. It is true that it thus escapes the very sense which was intended to act as a sentinel to the stomach; but it is none the less injurious, and is exceedingly likely to cause sickness, butyric acid being, as already mentioned, excessively irritating to the stomach. Another frequent cause of dyspepsia is want of cleanliness in the cooking utensils. The changes which such a want of cleanliness produces in the food prepared in them are probably of so subtle a nature as at present to elude the research of the chemist; but, unfortunately, they act only too readily upon the human organism, and I believe that many families suffer from dyspepsia simply because their cooks do not take proper pains to clean the pots and pans sufficiently soon after use to prevent decomposition of the fat used in cooking. Bad cooking is also responsible for many cases of dyspepsia in another way. When food is well cooked and appetising, the pleasure that is given to the palate excites reflexly the secretion of saliva, which does its part in digesting the starchy part of the food. Moreover, the increased amount of saliva stimulates the secretion from the stomach, and this in turn stimulates the intestines; so that the appetising character of the food is a great aid to rapid and thorough digestion. The nervous stimulus which appetising food affords to the digestive process is even more important than the chemical stimulus just mentioned, for Pawlow has found that the gastric and pancreatic secretions are greatly increased by appetising food, although it may pass out through an oesophageal fistula and never enter the stomach. It is probably in consequence of this that one so often finds that food apparently unsuitable both in quality

and quantity, but cooked in an appetising manner and eaten in pleasurable surroundings, is digested with ease by patients, when plain and simple food, apparently much more digestible but less pleasing to the palate, does not agree. Indeed, if food is badly cooked and unappetising, so far from making the mouth water and stimulating the digestive processes in the way just mentioned, it tends rather to cause disgust, and through the nervous system to stop secretion of gastric juice, and even to produce nausea. But even when foods are properly cooked they may disagree from their chemical composition, and give rise to acidity, flatulence, and other discomfort. Thus, oily fish, such as herring, mackerel, eels, and salmon, may disagree from their tendency to become decomposed in the stomach, and to give rise to irritating products.

It has already been said that fats undergo little or no digestion in the stomach, and that the proper place for their digestion is the intestines; but sometimes they may be split up in the stomach, probably by means of microbes, setting free fatty acids which are exceedingly irritating; allusion has previously been made to the excessively irritating properties of butyric acid. Many children are quite unable to eat hot mutton-fat without being sick, and yet the same children may be able to eat cold mutton-fat without harm. The probable explanation of this is that the hot mutton-fat undergoes a certain amount of decomposition in the stomach, and yields irritating products which cause sickness and vomiting; whereas the cold mutton-fat, being harder, is less changed until it arrives in the intestine, where it undergoes proper digestion. Raw vegetables, as a rule, do not irritate the stomach unless they have been swallowed without thorough mastication; but they very frequently occasion great development of flatulence in the intestines. Unripe fruit not infrequently causes irritation both in the stomach and intestines, because the cellulose of which it is chiefly composed is very indigestible, and is attacked very slowly, if at all, by the digestive juices; moreover, unripe fruits are often simply crushed between the teeth and swallowed, in pieces of considerable size, without undergoing thorough mastication. Over-ripe fruit, or rather decaying fruit, is productive of discomfort from a different cause: although soft, and thus not likely to occasion mechanical irritation, it frequently contains quantities of microbes, which give rise to decomposition, with the evolution of gas and production of irritating products which cause diarrhoea.

Drinks.—Beer is a common cause of dyspepsia, more especially if it be new and the fermentation not thoroughly completed. Such beer undergoes further fermentation in the stomach and intestines, gives rise to acute indigestion, and, if its use be long continued, to chronic catarrh.

Vinegar taken in large quantities leads to chronic dyspepsia with consequent emaciation, so that it is sometimes taken as a means of reducing obesity. It is a most dangerous remedy for this purpose, and I have seen its use by a well-nourished girl lead not only to emaciation, but to phthisis consequent upon malnutrition.

Acid wines (*vins aigres*) have a similar action, and, if used regularly,

tend to produce gastric catarrh. Spirits in excess are also most injurious; and whisky and water (or brandy and water) at night, by its irritating action on the empty stomach, is very likely to produce sickness and vomiting in the morning.

Tea is another article of diet which is apt to cause dyspepsia; partly by its action on the stomach itself and partly on the nervous system. An infusion of tea contains a good deal of tannin, which, if taken along with butcher's meat, tends to harden the fibre and render it less digestible. Common experience has led people to avoid taking butcher's meat and tea at the same time, and rather to take with tea farinaceous food and easily digested proteins, such as boiled eggs or fish. High teas, in which butcher's meat is taken in considerable quantity with tea as a beverage, are a frequent source of severe and persistent dyspepsia. The effect of the tea upon the digestion depends a good deal upon the quantity used, its temperature, the frequency with which it is taken, and the kind of tea employed. A very strong infusion, especially when the water is allowed to stand on the leaves for a long time, is most injurious; and if it be taken scalding hot the effects are still worse. Constant flatulence, pain in the stomach, and emaciation are frequently noticed in poor women who come as hospital patients. Many of them keep the teapot on the stove the whole day long, so that the tea is constantly stewing, and what they drink is rather a decoction than an infusion. It is best to allow the tea to stand for not more than three minutes upon the leaves and then to pour it off; and in some cases the plan recommended by the late Sir Andrew Clark of infusing the tea with milk instead of water seems to be very useful. If the water be very hard, effervescing water, such as soda or potash water, may be employed instead of well water. Some patients bear China tea better than the teas of India or Ceylon. Tea-tasters tell me that although all the tea plants come originally from China, yet the tea plants have been grown in the same soil for centuries in China; for about sixty or seventy years in Assam, and for about thirty years in Ceylon. But the Ceylon and Indian teas are really drunk much stronger than China teas; many people judge of the strength of an infusion entirely by its colour, and, as I have been informed by persons in the tea trade, the China teas yield more colouring matter than Ceylon or Assam teas, they contain with a good depth of colour less of the other ingredients than Assam and Ceylon teas. These latter teas should be drunk in a pale straw-coloured infusion instead of the dark-brown infusion of the China tea, as the pale infusion of the former corresponds in strength to a dark infusion of the latter.

The quantity of fluid taken at meals is often of importance. In the case of tea a chemical action of the liquid interferes with digestion; but even too much water with meals may cause dyspepsia. One reason of this is that if water be frequently sipped during the meal the food is apt to be softened in the mouth by water instead of by saliva, and thus passes into the stomach imperfectly masticated, and with its farinaceous ingredients little changed by the saliva. The fluid also

dilutes the gastric juice and renders digestion in the stomach slow, so that time is afforded for the growth of microbes, which is further favoured by the dilution of the gastric juice which in its normal concentration has an antiseptic action. Dyspeptics are frequently advised not to drink during meals, but to drink freely after they have finished their meal. This advice is good so far as it goes, because it prevents the patient from washing his food down without either mastication or insalivation; but it does not prevent dilution of the gastric juice. A better plan is to drink water, and especially hot water, an hour before meals (*vide* p. 369).

Microbes.—Imperfectly cleaned dental plates or carious teeth may form a breeding-ground for microbes which are carried down from the mouth into the stomach. Frequent swallowing of saliva certainly seems to give rise to dyspepsia occasionally, and possibly the dyspepsia which has been observed along with dirty dental plates may really have been due to profuse salivary secretion caused by the irritation they produce. Such secretion may also be induced by tobacco, either smoked or chewed, and this also leads to dyspepsia; while the action of the tobacco itself upon the nervous system is, in some cases, distinctly injurious; in others, however, if used in moderation, it may on the whole be beneficial. I have seen at least one case of dyspepsia in which everything failed to give relief until a naso-pharyngeal catarrh, from which the patient suffered, and which gave rise to constant swallowing of mucus, was treated by the removal of adenoids; after this the dyspepsia disappeared.

Nervous Influences.—The effect of emotion upon the stomach is well known. A piece of bad news takes away the appetite, and may even bring on sickness. Mental work immediately after a meal is very apt to disturb digestion, and if carried on regularly may lead to dyspepsia. I was once staying at a hydropathic establishment near a large commercial town where I observed that one of the rules was that any patient taking up a newspaper within an hour after dinner was fined one shilling. The reason for this was that the patients who came there were chiefly engaged in business, and the first thing they turned to in a newspaper was the money column; thus their minds became occupied with commercial affairs after meals and digestion was not so good. Robert Mayow put the case very well more than two hundred years ago. It was then supposed that all the functions of the body were carried on by vital spirits,—little imps which rushed hither and thither according to the work required in one part of the body or in another. He said that if the vital spirits are in the stomach digesting food they cannot be up in the brain, and, therefore, immediately after a heavy meal people are stupid and languid; if the vital spirits leave the stomach and go up to the brain, the digestion is not performed. If we put the word "blood" instead of vital spirits, Mayow's doctrine corresponds very nearly with that of modern physiologists. Excessive weariness tends to cause indigestion, because the wearied stomach and nervous system do not respond to the stimulus of

food. Worry, anxiety, disappointments, and especially love affairs, all tend to cause dyspepsia.

Exposure to *cold*, probably by disturbing the balance of the circulation, causes dyspepsia; and many people who are perfectly well during dry weather begin to suffer from dyspepsia as soon as the weather becomes cold and damp.

Want of Exercise.—General atony of the body, such as results from lack of exercise, brings about atony of the stomach as well; and the imperfect combustion or elimination of waste products not only lessens appetite but interferes with the digestive processes. Atony of the muscles of the abdominal wall renders them incapable of giving that support to the stomach and abdominal viscera which is necessary to keep them perfectly in position: the stomach and intestines, therefore, tend to fall, and the condition known as gastroptosis and enteroptosis results (*vide* art. "Visceroptosis," p. 860). In some cases these conditions are increased and perpetuated by adhesions of the omentum from old inflammation, especially about the caecum or appendix.

Adhesions of the intestines themselves also tend to interfere with the digestion, and give rise to flatulence, pain, and other discomfort. Pains which are probably due to old adhesions are sometimes very troublesome indeed (*vide* Vol. II. Part I. p. 951). The patient wakes early in the morning with pain in the abdomen of a weary, heavy kind, and this may continue for one or two hours. It may be relieved by the passage of flatus, and sometimes disappears entirely as soon as the patient gets up. In other instances the patient suffers from it while upright and is relieved by lying down. The pains are affected by exposure to cold, by changes of weather as well as by indiscretions of diet; and they seem really to be of a gouty or rheumatic origin, and may coexist with or alternate with headache or pains in the joints. (See also art. "Visceroptosis," p. 860.)

In some cases a *floating kidney* seems to produce no harm whatever; in others it causes constant dyspepsia, languor, depression, inability to work, and discomfort so great as to render life a burden to the patient. (*Vide* art. on "Movable Kidney," Vol. IV. Part I.)

There is one cause of dyspepsia which must be noted with great care, and this is incipient *phthisis*. There can be little doubt that chronic dyspepsia, by weakening the resistance of the organism, renders it less able to resist the attacks of microbes, and thus renders the individual more liable to phthisis; but, at the same time, the tuberculous disease in the lung itself appears to lead secondarily to dyspepsia. Whether this be due to a reflex action from the lung upon the stomach through the vagus, to toxins or toxalbumins, or to some other cause, I cannot say; but in all cases of dyspepsia, especially in patients from seventeen to twenty-five, the lungs should be carefully examined.

Consequences of Dyspepsia.—Patients suffering from dyspepsia are very apt to treat themselves, to cut off one article of diet after another, often without success, and in this way to lessen their nutrition and

diminish their strength without adding to their comfort. The constant irritation which they experience from discomfort or pain is apt to lessen their power of attending to other things; so that they lose interest in outside circumstances, have less power of attention, and are tempted to concentrate their thoughts upon themselves. When they attempt to read or think they feel dull and heavy, and they do not either comprehend so readily or remember so distinctly as they did when well. Moreover, they are likely to become irritable and snappish, and such fits of irritability often alternate with feelings of depression and languor. They sometimes also complain of singing in the ears and giddiness, and of a tendency to fall. These symptoms are more frequent in elderly people, and are probably associated to a considerable extent with atheroma. When they occur in younger persons free from organic disease they are more suggestive of excessive tobacco-smoking.

Treatment. In the treatment of dyspepsia it is important to lay down rules for the patient when food should be eaten, how food should be eaten, and what food should be eaten. The general experience of mankind shews that four or five hours should intervene between meals; but under certain conditions it is advisable to have something to eat every two hours. Baedeker's *Guide*, under general directions to travellers in the Alps, advises that something should be eaten every two hours in order to avoid the fatigue that might otherwise come on. In fever, in which the waste of the body is as great or even greater than in such violent exertion as Alpine climbing, every two hours is the time usually suitable for the administration of food; but when given so frequently as this it should be taken in small quantity; if much be taken at such short intervals indigestion will probably ensue.

How the Food should be Eaten.—It is evident, then, that food should be eaten slowly, and should be thoroughly masticated and insalivated before it is swallowed. I repeat that hurry at meals is a frequent cause of dyspepsia, and that hurry, worry, or mental exertion immediately after meals is another. In dyspeptics a certain amount of time should be allowed between the meal and the return to work, whether bodily or mental. Rest before meals is also an important factor, especially rest for a few minutes just before the evening meal, whether it be called dinner or supper; for this is the time at which the body in general and the stomach in particular are likely to be exhausted.

I have already said that much fluid at a meal, by diluting the saliva and gastric juice, is disadvantageous. It is not so injurious when taken immediately after a meal; but the best time to take liquid is an hour before meals, and the best form in which it can be taken is hot water. By drinking a tumbler of hot water an hour or so before meals, not only are the remnants of the previous meal washed out of the stomach, but also much of the water becomes absorbed. This prevents thirst being felt, obviates the necessity for drinking at meals, and, instead of the gastric juice being diluted by the water which would then be drunk, and digestion retarded, the water already absorbed supplies material for the

free secretion of saliva and gastric juice, and thus accelerates digestion. If the stomach be actually dilated, and especially if this be due to pyloric obstruction, the hot water will remain in the stomach for more than an hour after it has been drunk, and instead of accelerating digestion will interfere with it. In such cases the long interval which usually elapses between dinner, supper, and breakfast may allow the stomach to become empty, and breakfast may be eaten with relish and digested with comfort. But under such conditions a tumbler of water either hot or cold, taken on rising and retained, may prevent digestion and do harm instead of good.

The experience of mankind has shewn that it is advisable, to a certain extent, to separate the protein from the farinaceous meals; a very common plan is to have a farinaceous meal with a small quantity of protein for breakfast, a protein meal at luncheon, a very little farinaceous food in the afternoon, and again a protein meal in the evening. Thus, we find that at breakfast, bread and toast with some easily digested protein —such as egg or fish—is very commonly taken; or a small quantity of a sparingly digestible protein such as fried bacon, of which the hydrocarbonaceous fat frequently forms a large portion. At lunch the main part of the meal consists of meat, although a moderate quantity of bread or vegetables may be taken with it. The afternoon meal consists of a little bread and butter, and in the evening there follows another protein meal like that of lunch. In cases of dyspepsia it is often useful to separate the protein from the farinaceous foods more completely. The time of digestion of the two is different, and the parts of the digestive canal in which digestion takes place also differ.

For dyspeptics, a breakfast consisting of dry toast, rusk, or stale bread, with a little butter, which ought to be of the very best quality, should form the staple of the meal. If the digestion be very bad, the bread may have some hot milk poured upon it and be eaten in this way without butter; but ordinarily more milk, either warm or cold, may be taken with the toast, and many people can without injury take with the milk sufficient tea, coffee, or cocoa to flavour it. If the stomach be able to bear it, a soft-boiled egg or a piece of fish may be added. The mid-day meal should be chiefly protein, such as fish, fowl, eggs or meat, with some stale bread. One of the lightest fishes is boiled whiting, for the fibre is very soft and easily disintegrated. Cod is also good, but is somewhat harder and requires more careful mastication. Boiled or broiled sole is also excellent. If the sole be fried the skin must be very carefully removed as well as the bones. Sauces, containing fluid fats, are apt to disagree; and it is safer to take the fish simply with salt and stale bread. In many cases it is better rather to spread a little good butter upon the bread and take that with the fish than to use sauce or melted butter. In the same way fowl may be eaten with stale bread. In some cases eggs in the form of an omelette are tolerated, but in many patients eggs are prone to disagree. A cut from the joint, a chop, or a steak sometimes suits well, provided it be thoroughly masticated and all

the stringy parts avoided. It is best, as I have already said, to drink hot water an hour before lunch; but sometimes dyspeptics will not or cannot do this, and such persons may be allowed half a tumbler of water, which should be sipped after the lunch is over. In many cases it is advantageous to add to it one or two tablespoonfuls of old whisky or brandy. Effervescing water to many persons is more palatable and more stimulating than plain water; and it may be used provided it do not give rise to flatulent distension, as in many cases it does. As a rule, wines and beers do not agree so well with dyspeptics as spirits, but it must be borne in mind that as spirits tell less upon the stomach and more upon the liver, while wine and beer tell more upon the stomach and intestine, the consumption of the former is more liable to abuse than the latter; the patient who uses spirits is longer in finding out the mischief of excessive indulgence than he who uses wine or beer. In the afternoon hot water may be drunk again, flavoured by a piece of lemon floated upon it; and a small piece of bread or biscuit may be taken with it. Weak tea is more palatable, or milk and water, and if found not to disagree, either the weak tea, or milk flavoured with tea, may be used in the place of hot water. For the evening meal at seven or half-past seven similar food should be taken to that of the luncheon.

If the dyspepsia be less severe the patient may return to more ordinary diet, mixing the protein and farinaceous foods in larger quantity; but care must be taken by all dyspeptics to avoid too much sugar, which is very apt to create acidity. A good general rule also is to avoid all skins and bones, and all strings, stones, or seeds. The bones of fish, and the chips of bone that occasionally occur in curries or hash, are very indigestible; so are skins of every kind, whether they be skins of fish, flesh, fowl, fruit or vegetable. All stringy meats, fruits and vegetables should be avoided, and stones and seeds are wholly indigestible.

In regard to mastication an excellent rule is that of the late Sir Andrew Clark. The mouth contains, or ought to contain, 32 teeth, and to every mouthful of food 32 bites should be given. If the teeth are imperfect, even this number is not sufficient; and as many as 64 or 96 bites may be required properly to comminute a single mouthful of steak or chop. Another rule, which is sometimes of great service in dyspepsia, is to insist that before the food is swallowed it should be so finely masticated, and so thoroughly mixed with saliva, that it shall be of the consistence of cream, and would pass through a sieve without leaving remainder. It is very much easier, however, to give such a rule than to get it followed; and it is only in very severe cases, and especially in those in which the pain is so great as to suggest the presence of gastric ulcer, that patients will follow it. They often find the rule so troublesome that mere inconvenience or discomfort will not force them to follow it: and they can only be induced to do so by finding that obedience to it prevents the occurrence of severe pain, while the neglect of it causes the pain to return.

Warmth and Support. Regimen.—Dyspepsia often results from undue exposure. There are four places that require special warmth, namely, the back of the neck, the front of the abdomen, the shins, and the feet. Many persons, after sitting in the draught which always exists in cold weather between the door and the fireplace, will suffer from dyspepsia, and then wonder what article of food has disagreed with them. This draught should be carefully avoided by raising the feet above its level, or by sitting out of the current from the door to the fire. Thick boots, especially boots with cork soles, woollen stockings, and gaiters of cloth or leather protect the feet and legs from cold; and warmth to the back of the neck may be afforded by a muffler or high collar. Another aid of great importance in cases of dyspepsia is an apron of chamois leather and flannel or a belt round the abdomen, which should be worn in such a way as to give both warmth and support; support is especially needful in cases in which the abdominal muscles are lax and the belly pendulous. The commonest kind of belt is either flannel or knitted woollen; but the silk scarf known under the name of "cummerbund" is even more comfortable. It should be long enough to go three times round the body, and it can be put on with any degree of tightness. In cases of floating kidney the belt may be provided with a pad or truss (*vide* p. 881).

It is important, as far as possible, to avoid *fatigue* before meals and to get rid of all *worry* or thoughts about business during meals. Some people damage their digestion by walking home from their work with the notion of getting an appetite. The extra labour caused by this finishes up the patient already exhausted by his daily work, and lessens the digestive powers still more. Twenty minutes' rest at least, after getting home, is a useful restorative; and is advisable for dyspeptics, especially for patients at or above middle age. On the other hand, if the occupation be of a harassing and anxious kind, it is sometimes useful for the patient to walk home instead of driving in the hope of getting rid of his anxiety and worry by exercise. If rest can be taken on arrival the disadvantage caused by the extra bodily work in such cases may be more than compensated by the relief to the mind.

After the meal is over, rest is required both for body and mind; and active exertion, either bodily or mental, is injurious; it is advisable to rest half an hour or more if possible. During this time pleasant conversation or light reading or a pipe may divert the thoughts from care.

The *medicines* that have been used in the treatment of dyspepsia are almost innumerable; but they may be divided into a few classes: (i.) those which stimulate the secretion and movements of the stomach; (ii.) those which by their local action have a sedative action on the stomach; (iii.) those which act upon the general nervous system; (iv.) those which supply digestive material; (v.) those which lessen abnormal decomposition; and (vi.) those which aid in elimination. Among those which stimulate the stomach directly one of the best is bicarbonate of soda in 10 or 15 grain doses. This should be given 15, 20, or 30 minutes before meals; and it may, I think, be combined

advantageously with some bitter, such as calumba, quassia, gentian, or camomile. Another exceedingly useful stimulant is rhubarb. An old-fashioned plan was to make the patient chew a stick of rhubarb, in which way he got a solution of its active principles in the alkaline saliva; but many persons dislike this way, and prefer the rhubarb taken along with an alkali before meals. A very good remedy is the compound rhubarb powder of the British Pharmacopoeia—long known as "Gregory's Powder"—in doses of 5 or 10 grains in a cachet with a mouthful of water 20 minutes before meals. Another very useful mixture is the following, which is much used at St. Bartholomew's Hospital:—℞ Sodii bicarbonat. gr. x., tinct. gentian. co. ℥xxx., spt. chloroform. ℥x., infus. rhei ʒss., aq. menth. pip. ad ʒj. M. F. hst. mitte doses viii. Sig. Two tablespoonfuls to be taken three times a day twenty minutes before meals. I have already made a comparison between itching of the skin and irritation of the stomach; and chrysophanic acid, so useful in skin diseases, appears also to have a most useful effect in disease of the stomach. One of the best combinations that I know is one which was a great favourite of the late Dr. Warburton Begbie; its effects were so good that he used to call it the Pulvis mirabilis:—R Bismuth. subnit. vel bismuth. carb. gr. v., sodii bicarb. gr. v., pulv. rhei gr. i., pulv. nuc. vom. gr. ss., pulv. cinnamom. co. gr. iss. M. The powder to be dispensed in a cachet; two cachets to be taken with a mouthful of water three times a day twenty minutes before meals. This powder may also be given in cachets containing 10 grains each, and the quantities of the several ingredients may be increased or diminished; so that if the quantity of rhubarb in the above formula cause the bowels to move too freely it may be lessened as required.

A great deal has been written about the uselessness of bitters, and perhaps their utility has been exaggerated; nevertheless in many cases they certainly seem to be productive of great benefit. In cases of atonic dyspepsia, such as we see ordinarily in hospital patients who complain of windy spasm and present a certain definite group of symptoms, quassia and iron before meals appear frequently to be more beneficial than alkalis. The group of symptoms consists in a pale flabby tongue, furred on the dorsum, and marked with teeth at the edges; pain in the epigastrium striking through between the bladebones; much wind in the stomach; flushes of heat; black specks before the eyes, and pain at the top of the head. If constipation be present, some sulphate of magnesia in the morning greatly assists the action of the iron and bitter.

In irritable dyspepsia bismuth is one of our most useful remedies; it may be given in the form either of a solution or of a powder. Some patients object to a powder, and prefer the liquor bismuthi, which may be given in doses of half a drachm to a drachm with aromatic spirit of ammonia and some carminative water. The subnitrate of bismuth does not go very well with bicarbonate of soda, as decomposition occurs with the formation of carbonate of bismuth, nitrate of soda, and evolution of carbonic acid gas, which, if the quantity of bismuth be large, may be so

great as either to blow the cork out of the bottle or even to burst the bottle itself. In books on materia medica it is the fashion to recommend the carbonate or submitrate of bismuth to be suspended by means of mucilage or compound powder of tragacanth; but many patients dislike this thick draught, and prefer the powder simply shaken up in the vehicle. I have already given a formula for bismuth in a cachet with soda. When much pain is associated with taking of food, 20 to 30 minims of the compound tincture of camphor may be added to the bismuth mixture with great advantage. When there is great pain and acidity after meals, it may be relieved by dissolving a teaspoonful of bicarbonate of soda in a little water, and sipping this with a salt-spoon until the pain is relieved; this is, of course, but a palliative measure, yet it gives considerable relief at the time, and (although it is often said to destroy the coats of the stomach) I have known cases in which the practice had been continued for years without any apparent injury to the patient.

Among the drugs which assist the function of digestion through the nervous system, the foremost place must be accorded to nux vomica and strychnine; and 10 minims of the tincture of nux vomica, or 5 of the liquor strychninae, are most useful adjuncts to antidyspeptic remedies in most cases of feeble digestion. Among those substances which are used to supply digesting material the most important are hydrochloric acid and pepsin; to these perhaps rennet should be added. A dose of 10 minims of dilute hydrochloric acid or of nitro-hydrochloric acid appears to help digestion in many cases in which the acid only is deficient. It is frequently given with the bitter shortly before meals, as then it seems to stimulate the appetite; but it may be given also immediately after meals, either alone, with tincture of nux vomica, or with pepsin. In some cases the gastric juice appears to be deficient in rennet. The exact use of this ferment in ordinary digestion has not yet been ascertained; but that it has some important function I am convinced, for in making some experiments a good many years ago on the digestive power of various preparations of pepsin, I found one which contained hardly any pepsin but a good deal of rennet; yet, contrary to expectation, it was of considerable benefit to some of my patients. Ingluvin in some cases, especially where there is a good deal of vomiting, is even preferable to pepsin.

When farinaceous food is badly borne and gives rise to gastric flatulence, diastase, given either with or before a meal, is useful, and pancreatic preparations may sometimes do good also, although they may interfere with the pepsin unless the contents of the stomach are acid. As a rule preparations of pancreas should be given two or three hours after a meal when the food is leaving the stomach, and if there is hyper-acidity an alkali should be given at the same time.

Amongst the remedies which tend to prevent fermentation, one of the best is phenol, which may be given in the form of a pill, half a grain to a grain with every meal. Creasote has a similar action, and both it and phenol

may be given in doses of 1 to 3 minims, along with oil, in the form of capsules or perles. Sulpho-carbolate of soda in 10-grain doses is also a very efficient remedy. For preventing decomposition in the intestines and flatulent distension, besides the remedies already mentioned, salicylate of bismuth, with salol, β-naphthol, or with salicylic acid, is often of great use. Cyllin in doses of 1 to 5 minims may be used both for flatulence in the stomach and intestine. Three minims is a convenient dose in a capsule made either of gelatin so as to dissolve in the stomach in gastric flatulence, or of keratin, which will pass through the stomach and dissolve in the bowel when an intestinal disinfectant is desired. Charcoal in the form of biscuits, powder, or granules is sometimes very useful in lessening flatulence, either gastric or intestinal. The accumulation of faecal matter in the intestine must be prevented and the bowels kept regular by means of purgatives (*vide* "Constipation," p. 648). The evacuation of the liver is also a most important matter. The mode of action of mercury upon the liver has not yet been fully ascertained; but there can be no doubt whatever that a dose of blue pill or calomel at night, followed by a saline purgative in the morning, is of the greatest use in relieving many of the symptoms of dyspepsia. In some patients mercurials followed by a saline cause great depression; but this depression may be relieved, without bringing back the dyspeptic condition, by a small glass of champagne or by two tablespoonfuls of brandy in a glass of soda water.

The pains to which I have alluded as probably dependent upon adhesion of the intestine, and occurring in gouty or rheumatic subjects, are best treated by salicylate of soda and salicylate of bismuth with warmth to the stomach and support by means of a belt. Although they may be increased by irritating articles of diet, such as nuts, figs, and like indigestible substances, yet diet does not exercise nearly so much influence over them as exposure to cold, indulgence in acid wines, or in any other excess that usually brings on gout or rheumatism.

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L. B.

Dyspepsia in Childhood.—Disorders of digestion occurring in children below the age of puberty differ in several important particulars from those encountered at a later age. In the first place, the deficient development of the stomach in early life necessitates the greater part of

the digestive functions being carried on by the intestine: consequently any failure of assimilation of the food usually manifests itself by symptoms which are intestinal rather than gastric. In the second place, children are very susceptible to catarrhal affections of the gastro-intestinal tract, the symptoms of which are apt to be obscured by phenomena arising from reflex irritation of the nervous system. Chronic inflammation of the stomach, on the other hand, which constitutes one of the commonest causes of dyspepsia in adult life, is rare in young children. Finally, functional disorders of the stomach associated with an abnormal secretion of gastric juice (hyperacidity, hypersecretion) are very rare below the age of puberty.

A. Gastric Dyspepsia.—1. **Weak Digestion.**—This name is given to a functional disorder of the digestive organs characterised by failure of the secretory and motor powers of the stomach and intestine.

Etiology.—In some cases a weak habit of digestion occurs as a primary complaint, but in the vast majority it ensues upon the damage inflicted upon the mucous membrane of the alimentary canal by an attack of catarrhal inflammation. In 38 per cent of the cases of primary dyspepsia which have come under my own care, either one of the parents, or other children in the family, had suffered in a similar manner.

Predisposition to the complaint is more often transmitted through the mother than the father, and is more common in girls than in boys. The children of neurotic parents are particularly apt to fall victims to the disorder, as are also the offspring of persons who have married late in life. Among the exciting causes of weak digestion, irregularity of feeding and over-indulgence in rich or indigestible substances rank first. In rarer instances the disorder can be traced to overcrowding, deficient ventilation, septic states of the mouth arising from stomatitis or carious teeth, faecal accumulations in the lower bowel, a neglected hernia, or excessive masturbation. When the complaint is due to antecedent inflammation of the alimentary tract it usually begins between the ages of two and five, and frequently persists throughout life. Occasionally the disease dates from an attack of some infectious disorder, such as scarlatina, measles, small-pox, or pertussis, the symptoms first becoming noticeable during the period of convalescence.

Symptoms.—Pain after food is one of the most prominent features of the complaint. It usually begins shortly after a meal, and is chiefly experienced between the shoulders or behind the lower end of the sternum. Occasionally the skin over the lower ribs on the left side feels bruised and is tender to the touch. Flatulent eructations occur after each meal, and may be excited even by a draught of water or a cup of tea. Towards evening the stomach and intestines often become distended with gas, and the pressure which they exert upon the diaphragm gives rise to an inspiratory form of dyspnoea and palpitation. Nausea is a frequent symptom of the complaint, and may persist for many days or even weeks, being temporarily relieved by food and stimulants. The appetite is usually variable, being at one time ravenous and almost insatiable, while

at another it can be easily appeased or is altogether absent. The tongue is large, pale, flabby, and often indented along its margins by the teeth. The bowels are usually sluggish in their action, and the stools hard and pale, or putty-like in consistence, but attacks of diarrhoea are apt to supervene from time to time, when the motions not infrequently consist almost entirely of mucus. The pulse is soft, small, and during the period of digestion abnormally slow. The urine is diminished in quantity, neutral or alkaline in reaction, and deposits phosphates on standing. Vomiting, except as the result of an unusually copious meal, is an inconstant feature of the complaint. Some children, however, habitually vomit solid food, such as meat and fish, whenever they are permitted to partake of it. Although actual emaciation does not usually accompany this condition of feeble digestive power, the child fails to thrive, and becomes dull, spiritless, and disinclined to the least physical or mental exertion. Many patients suffer from extreme languor and drowsiness after meals, or invariably sigh, yawn, or hiccup during the course of the afternoon. Occasionally at these times a strange confusion of ideas may be observed, or the child may stammer or become partially aphasic. In other cases it exhibits various choreic movements, such as nodding the head, blinking the eyes, or incessantly twitching the extremities. In some cases severe frontal headache, which comes on before breakfast and persists until the afternoon, is the chief source of complaint. Among the secondary phenomena of the disease attacks of asthma, palpitation, giddiness, faintness, incontinence of urine, and irritable skin eruptions are the most important. The asthmatic attacks (asthma dyspepticum) appear suddenly after meals, and are characterised by urgent dyspnoea, cyanosis, and a slow or irregular pulse. These symptoms rapidly subside after vomiting has taken place. Among the cutaneous affections eczema and urticaria are the most common. The former is especially apt to shew itself after indulgence in such articles as porridge, strawberries, fish, almonds, pork, and cucumber; while the latter appears chiefly at night-time upon the back, buttocks, and thighs. When the contents of the stomach are examined after a test-meal the total acidity is found to be diminished, and the percentage of hydrochloric acid considerably below the normal. From the weak state of the wall of the stomach the period of gastric digestion is much delayed, and fermentation of the stagnant food is apt to occur.

An important variety of enfeebled digestion accompanies general neurasthenia. This complaint is chiefly met with in boys between the ages of ten and sixteen, and can frequently be traced to masturbation. Pain, flatulence, and distension occur immediately after each meal, the bowels are obstinately confined, and the appetite is deficient. Constant headache, a sense of extreme physical exhaustion, inability to concentrate the thoughts, anaemia, and emaciation are prominent symptoms. The disorder runs its course in about three months, and if treated by rest in bed, massage, and a milk diet, does not necessarily lead to any serious disturbance of the digestion in later life. Relapses, however, are not infrequent.

Treatment.—The general principles of treatment are identical with those observed in the atonic dyspepsia of later life. The clothing should be warm, and regular exercise must be taken every day in the open air. Excess of fluid with the meals must be avoided, and each mouthful of food must be well masticated before being swallowed. Green vegetables should be given sparingly, and only a small quantity of good potato allowed with the midday meal. No food should be permitted after six o'clock in the evening. The first indication for medical treatment is the due regulation of the bowels. When a complete evacuation has been secured by means of castor oil or an enema, a small dose of cascara combined with maltine may be given twice a day after meals, or some simple aperient may be prescribed each night. As soon as the bowels act regularly the administration of gastric antiseptics may be begun; of these carbolic acid and resorcin are the most suitable, and may be given, with bicarbonate of sodium, two or three times a day, about two hours after food. When the abdominal distension and flatulence have quite subsided, measures should be taken to stimulate the functions of the stomach. In young children the mixture of rhubarb and soda administered before the meals is usually of great value; but at a later age hydrochloric acid and pepsin are to be preferred. All tonic remedies must be used with caution owing to their tendency to excite gastric catarrh. When, however, the gastric symptoms have quite subsided, a careful trial may be made of the milder preparations of iron and cod-liver oil; but in all cases a dose of mercury and chalk or blue pill should be given once or twice a week during the continuance of the tonic treatment.

2. **Acute Gastric Catarrh.**—*Etiology.*—Gastric catarrh of an acute or subacute character may occur either as a primary disease, or as a complication of some other and graver condition. The *primary* variety is extremely common in feeble and debilitated subjects, and its inception is favoured by such constitutional diseases as tuberculosis, anaemia, rickets, and congenital syphilis. Under such conditions an attack is often directly excited by exposure to wet and cold, especially during the spring and autumn months; but more commonly the cause is to be found in the administration of unduly rich or indigestible foods. It may also be mentioned that acute gastro-intestinal catarrh often manifests itself in an epidemic form in localities where enteric fever is prevalent, and is due to the use of contaminated milk or drinking-water. Gastric catarrh of *secondary* origin is much less common in children than in adults. It chiefly occurs during the early stages of such specific fevers as scarlatina, measles, erysipelas, and influenza; or as the result of chronic diseases of the heart, lungs, liver, or kidneys. Occasionally the disease is accompanied by the formation of a false membrane, as in the form of gastritis that sometimes complicates diphtheria.

Clinical Varieties and their Symptoms.—Owing to the numerous ways in which gastric catarrh may arise, and to the uniformity in the general symptoms of the disease, whatever its exciting cause may be, it is found expedient to divide the complaint into two principal varieties,

called respectively the "febrile" and the "non-febrile," according as the temperature of the body is raised or not. (i.) Febrile gastric catarrh, sometimes known as "gastric fever," is a very common complaint in delicate children between three and twelve years of age. It usually begins with shivering, headache, and pains in the limbs; but occasionally its onset is accompanied by convulsions. The temperature rises rapidly, and may attain 103° F. by evening, when the child is apt to become slightly delirious. Vomiting is an inconstant symptom, but in some cases repeated emesis occurs, with the ejection of bile and mucus. The bowels are usually confined, but if the catarrhal affection spread to the mucous membrane of the intestines the child may have colicky pains in the abdomen accompanied by a watery and offensive diarrhoea. When the duodenum is inflamed jaundice may result. The tongue at first is thickly coated upon the dorsum, while its tip and edges are clean and red. In severe instances the organ becomes dry and fissured, the lips parched and cracked, and sordes appear on the teeth. The appetite is completely lost and thirst is excessive. Frontal headache, restlessness, and mental depression continue throughout the attack. Sleep is disturbed, and nocturnal delirium may be present. The pulse is quick, weak, and compressible, and the urine is high-coloured, and passed in diminished quantity. The fever is markedly remittent in character, the temperature rising to 101°-103° F. at night, and falling to 99°-100° F. in the morning. It attains its maximum about the third day, after which it gradually declines, becoming intermittent, and finally subnormal. Occasionally the disease ends by a veritable crisis. As a rule an attack lasts from seven to ten days; but if the stomach be irritated by the administration of stimulants or tonics, it may continue for a much longer period. When the disease occurs in an epidemic form the temperature often remains high for ten days or a fortnight, and abdominal pain with diarrhoea is usually present. It subsides in a gradual manner, and may be followed by one or more relapses. The disorder is distinguished from enteric fever by its sudden onset and shorter course, and by the absence of tympanites, enlarged spleen, and of the characteristic eruption.

(ii.) Non-febrile gastric catarrh.—There are three clinical varieties of this disease which are worthy of special notice, namely, acute indigestion, subacute gastric catarrh, and recurrent gastric catarrh.

(*a*) Acute indigestion, or as it is often called, "embarras gastrique," is most frequent after the age of five years. The symptoms begin suddenly within a few hours of a meal, or during the night; they consist of pain or discomfort at the epigastrium, flatulence, nausea, and an excessive flow of saliva. After a variable period of time vomiting occurs, and the stomach rejects the greater portion of the last meal in an undigested state. Although the evacuation of the stomach usually affords relief, the nausea soon returns, and may be accompanied by a sense of faintness or by violent palpitation. The pulse is weak, fluttering, and often abnormally slow; the face is pale, the eyes are sunken and surrounded by dark rings, and the forehead and extremities are bathed

in perspiration. These symptoms culminate in violent retching and vomiting, which recur at short intervals, and end in the expulsion of small quantities of bile-stained mucus. The appetite falls into complete abeyance, and any attempt to take food or even water only aggravates the retching. In some cases the irritant contents of the stomach find their way into the intestine and set up acute diarrhoea. When improvement sets in the attacks of emesis gradually become less frequent, and finally the child falls into a heavy sleep from which it awakes free from nausea. In rare cases an attack of acute indigestion is ushered in by aphasia, asthma, or a series of epileptiform convulsions, which subside immediately the stomach rejects its noxious contents. The disorder usually runs its course in twenty-four to forty-eight hours, but it is apt to be followed by general malaise, headache, laryngeal catarrh, or bronchitis.

(β) Subacute catarrh of the stomach, popularly known as "biliousness," is a very common complaint between the ages of four and sixteen. The subjects of this disorder suffer from chronic depression of spirits, and are listless and apathetic in their movements. They usually present a pale or sallow complexion, with dark lines beneath the eyes, and a slight icteric tinge in the conjunctivae. In some instances the speech is hesitating or stammering, while in others the patient acquires a habit of frowning, winking the eyes, or making sudden grimaces. The temper is uncertain and peevish, while at night the sleep is disturbed by horrible dreams. In every instance constant and severe headache is a marked feature of the complaint. The pain is usually confined to the frontal region, but it sometimes radiates along the supraorbital nerves. It is most severe in the early morning, or after the midday meal, and is increased by active movements of the head, by stooping the body, and by the use of alcohol. Epigastric discomfort is experienced after meals, and towards evening the intestines become distended with gas. Nausea is common, but vomiting only occurs at intervals or after an unusually full meal. The appetite is much diminished, and the child often expresses the utmost loathing for fat and sweet foods. In the early morning the tongue is usually coated with a yellowish-white fur, but towards midday it may become clean and abnormally moist. Occasionally it is the seat of a symmetrical form of superficial ulceration due to exfoliation of the epithelium. Heartburn and eructations of fetid gas are a source of frequent complaint after meals, and the patient often yawns, gasps, or suffers from hiccup or "goose-skin" during the periods of digestion. The bowels are confined, and the stools are clay-coloured and extremely offensive. The pulse is small and feeble, the hands and feet perspire and exhale an unpleasant odour, and attacks of syncope are apt to occur from time to time. The duration of the disease is uncertain. When appropriately treated it usually comes to an end in a few days, but when neglected it may be prolonged for many weeks.

(γ) *Recurrent Gastric Catarrh.*—This disorder, which has been variously described by the terms "periodic," "fitful," and "cyclical" vomiting, is

characterised by severe attacks of emesis which exhibit a tendency to periodic recurrence. The complaint usually begins between the ages of two and five, and according to my experience is more common in boys than in girls. An attack is often precipitated by exposure to cold, a change of diet, physical or mental fatigue, or excitement.

Although the attacks exhibit a certain periodicity they do not recur at regular intervals; for at one time several weeks or months may elapse between the consecutive attacks, while at another the disorder may recur once or twice a week. The most severe and obstinate cases are usually those in which the first symptoms date from the convalescent period of measles or typhoid. With the approach of puberty the incidence of the disease becomes less and less frequent, and finally it disappears altogether. In its clinical aspect this variety of gastric catarrh does not materially differ from acute indigestion. As a rule nausea and vomiting are the first symptoms to attract attention, but occasionally severe headache comes on before the emesis. The first effect of the vomiting is to rid the stomach of the remains of the last meal, the constituents of which shew few signs of digestion. Severe retching and vomiting recur at short intervals, and the ejecta soon come to consist entirely of thick mucus, or even of bilious fluid. During the course of the disorder the tongue presents a coating of white fur, the bowels are confined, and the face is pale and anxious. The urine is scanty, and may contain diacetic acid. The temperature of the body is subnormal. The duration of the complaint seldom exceeds twenty-four hours. Recovery is usually rapid, but when the attacks recur at short intervals the child becomes thin, anaemic, and irritable, and presents the symptoms of weak digestion.

Treatment.—In all inflammatory affections of the stomach and bowels rest in the recumbent posture is an imperative necessity. If the abdominal pain be severe the epigastric region should be covered with a large linseed poultice, or repeatedly fomented with hot flannels. When convalescence has set in the child may be sent to some inland health resort, like Malvern or Ilkley, but sea-air should be avoided. As long as the vomiting continues no attempt should be made to administer food by the mouth, but small pieces of ice may be sucked to relieve the thirst. When the more urgent symptoms have subsided, a cautious trial can be made of small doses of milk and soda-water, cold bovril, veal or chicken broth. In the febrile form of the disorder a liquid diet should be strictly enforced until the temperature begins to intermit, after which time milk puddings, bread and milk, or toast may be allowed. To prevent a recurrence of the complaint the diet should be strictly regulated to the exclusion of those articles of food that are prone to undergo fermentation in the stomach. Indulgence in sweets and cakes between meals must be prohibited, and the child should be taught to spend a certain time over each meal, and to chew each mouthful of food well before swallowing it. If the case be seen at the beginning of an attack, the stomach should be emptied by means of a full dose of ipecacuanha wine or sulphate of zinc: but if emesis has already occurred the child

may be encouraged to drink one or two tumblerfuls of warm water. As soon as the organ has been completely evacuated a few grains of calomel or mercury and chalk may be administered, and followed after a few hours by a full dose of effervescing magnesia or sulphate of sodium. Castor oil is also a valuable remedy at this stage of the complaint, but is apt to excite vomiting. Should the retching continue severe, and signs of exhaustion manifest themselves, it may be necessary to administer a minute dose of morphine, or a sedative enema. In the febrile complaint, in which the gastric symptoms are seldom of much importance, repeated doses of one-sixth of a grain of calomel, with the occasional use of an effervescing alkaline mixture, are usually sufficient. In the recurrent form of gastric catarrh a dose of the sulphate and carbonate of magnesia every morning before breakfast seldom fails to effect a cure. Edsall recommends alkalis for a similar purpose.

Acute Hypersecretion.—This disorder is supposed to be almost entirely confined to adult life, but it is highly probable that the complaint described by Rossbach in 1884 by the title "Nervous Gastroxynsis" as well as that denominated "Paroxysmal Hyperacidity" by the writer in 1896 are really examples of this morbid condition. In the majority of the cases which have come under observation up to the present time, the gastric symptoms have arisen without obvious cause, and have presented a remarkable tendency to periodic recurrence; but occasionally they have come on as a result of severe irritation in some organ situated more or less remotely from the stomach. Thus, I have several times observed characteristic attacks of acute hypersecretion in young girls immediately prior to the onset of the catamenia, and in one instance the vomiting of large quantities of gastric juice accompanied the passage of a small uric-acid stone down the ureter. I have also noted acute hypersecretion at the onset of Henoch's purpura. But whether the complaint arises in an idiopathic manner or ensues as a result of some other morbid condition, the symptoms by which it is portrayed are essentially the same.

Usually about midday or during the early afternoon the child is attacked by severe headache, which is markedly increased by any movement of the head, coughing, or sneezing; it is also worse if the child attempt to stand or walk. Occasionally the pain is so severe that the patient cries out at intervals as though suffering from acute meningitis, or he may become partially aphasic or even slightly delirious. After a variable interval violent pain is experienced in the stomach, and the legs are drawn up and the hands clasped over the abdomen in the effort to secure relief. Flatulent distension of the stomach and occasional eructations of gas make their appearance, and are followed by pyrosis and a scalding pain behind the sternum. Nausea and giddiness soon supervene, and the child suddenly sits up and empties the stomach without apparent effort. In some instances, the act of emesis is followed at once by the disappearance both of the headache and the gastric pain.

so that the patient will fall asleep and awake a few hours later quite restored to health. As a rule, however, vomiting recurs at short intervals, and several days may elapse before convalescence is established. Examination of the vomit shews that it consists entirely of a pale green or yellow fluid which contains a slight excess of free hydrochloric acid, and is capable of digesting egg-albumin; but towards the end of the attack the reaction often becomes alkaline from an excess of bile and mucus. The temperature of the body is usually elevated at the onset of the headache, but it subsequently falls below the normal point.

The main points in the *treatment* are to allay the pain and vomiting, and to prevent a recurrence of the disorder. Rest in bed is essential, and the child should be encouraged to drink Vichy or Contréxeville water, or warm water containing about two grains of bicarbonate of sodium to the ounce, with the view of neutralising the abnormally acid contents of the stomach. In severe cases it may be necessary to wash out the stomach with a mild alkaline solution, in order to allay the vomiting, and to support the general nutrition by enemas of peptonised milk. To prevent a recurrence of the complaint excitement and fatigue must be forbidden, and a dose of phosphate of sodium or sulphate of magnesium should be administered each morning before breakfast. Digestive troubles in the intervals between the attacks are best combated by a mixture of carbonate of bismuth, bicarbonate of sodium, and glycerin. Green vegetables must be given sparingly, and fruit entirely prohibited.

B. Intestinal Dyspepsia.—In addition to the various forms of intestinal indigestion which accompany inflammation of the bowel, and diseases of the liver and pancreas, children are apt to suffer from a curious array of symptoms which appear to depend upon a functional disorder of the large intestine (colonic dyspepsia). Most of the subjects of this disorder either belong to a tuberculous family or present chronic enlargement of the glands, hypertrophy of the tonsils, or adenoids. The complaint usually begins between the ages of five and ten, and is slightly more frequent in girls than in boys.

Symptoms.—Spasmodic pain in the abdomen is the principal symptom of the disease. It usually begins suddenly during the evening or in the early morning before breakfast; or perhaps about midday. Sometimes an attack is excited by physical fatigue, or by the use of indigestible foods. The pain is usually referred to the umbilical region, and often travels across the abdomen from right to left. Occasionally it is chiefly felt in the region of the hepatic or splenic flexures of the colon. It may prove severe from the very outset, but it generally grows more and more intense until a maximum is reached, when it afterwards gradually declines. During the painful crisis the face is pale and covered with sweat, and the child may roll about the floor or bed and scream with agony. Pressure over the abdomen usually affords relief. The appetite is extremely capricious, and a marked distaste is shewn for fat and sweets. Acid drinks and lemon-juice, on the other hand, are much

liked. Thirst is constant, and is apt to be excessive during the night. Constipation exists in about 70 per cent of the cases, but occasionally a lienteric form of diarrhœa is present, the bowels being moved immediately after each meal. Nausea and vomiting are seldom complained of, and the tongue is clean, red, and moist. The victims of this disorder remain thin and anæmic for years, and are prone to gastric or intestinal catarrh from slight causes. After puberty the various symptoms usually subside, but the tendency to constipation continues, and not infrequently an obstinate form of atonic dyspepsia persists for years.

Treatment.—All articles which contain a large percentage of indigestible material must be avoided, hence green vegetables should be given sparingly or withheld altogether. White fish, chicken, and tender meat are to be preferred to the coarser or richer varieties, and all condiments, curries, and highly spiced foods should be forbidden; milk and cocoa may be given freely, but coffee and strong tea should be prohibited. The meals must be taken at regular hours, and a basin of bread and milk may be allowed at bed-time. The first point that requires attention in the medicinal treatment of the disease is the regulation of the bowels. This is most readily effected by the administration of the liquid extract of cascara sagrada combined with extract of malt immediately after the morning and evening meals. Occasionally small doses of aloin and nux vomica answer well; or some simple laxative, such as the confection of senna and sulphur, may be employed with advantage. When the bowels are moved at each meal a small dose of morphine in an aromatic mixture should be given before the food. As soon as the bowels have been brought into a satisfactory state, one of the bland preparations of iron combined with belladonna will be found of the greatest benefit in preventing a recurrence of the abdominal attacks. Cod-liver oil should not be given until the intestinal pain has quite subsided.

W. Soltau Fenwick.

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NEUROSES OF THE STOMACH

AND OF OTHER PARTS OF THE ABDOMEN

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Introduction. — In the multiform perturbations of the stomach and other viscera the nervous system is often primarily concerned, and can never be a negligible factor. This factor in causation may indeed be over-estimated; or at any rate insufficiently correlated with other factors; yet Dr. Goodhart, in his lectures "On Common Neuroses," says of indigestion: "If I were going to write a book on indigestion I should first devote myself to a volume on diseases of the nervous system"; and again, "It is no great exaggeration to say that there are only two forms of indigestion: that produced by over-eating and drinking, and that due to a failure of nervous power." Nevertheless we must not be too ready to attribute to "visceral neuroses" pains and other distresses which may be due to local causes. The only way of safety in approaching a diagnosis of mere neurosis in gastric and other visceral sufferings is to make this the last of our alternatives, and to hold it lightly and critically.

In the integration of animal function the proper activities of all our organs have been so completely reduced to the governance of the nervous system, that in a sense it may be said that their every act, their every disorder, whether in defect or in excess, is a function of the nervous system. The whole store of the nervous system may run low; or excessive demand in one part of the body may divert a part of the due supply of nervous energy from another part, as from the stomach. No organ lives on its own funds; the very existence of a nervous system means a give and take between the several organs of the body: now one is in full play, now another; and this alternation of departmental activities illustrates the principle of greatest economy. Energy which in a resting organ might be running to waste is diverted to the uses of others in tides of activity. Thus the several organs are members one of another; when one suffers, another sympathises: and in clinical medicine it is one of our most interesting studies to discover these intimacies, to detect not only the general dependence of each organ upon the whole system, but also the closer alliances between groups of two or more organs. To some extent, no doubt, the nervous system is the fly-wheel of the whole machine; by its storage and distribution the various pulsations of the several parts are integrated: but it seems likely that within this great integration there are subordinate alliances of particular organs, so that a disturbance of one of them is felt at first rather by allied parts than universally. Broadly speaking, for instance, the organs of the circulatory,

of the alimentary, and of the respiratory systems hang together in special dependences; though when one or more members of any one of these systems or sub-systems is affected gravely, it will not be long before its defects are felt beyond its own circle. Strictly speaking, it is scarcely possible for any part to fail or suffer without some echo throughout the microcosm; in a sense, then, there can be no dyspepsia which is not associated with nervous affection, whether directly by way of the nerves themselves, or by way of intermediate intoxication spread by the blood to the nervous parts: these parts then suffer disturbance within their own sphere, and by their connexion with the stomach may intensify the original disorder, or, on the other hand, may bring about some conservative reactions of which we know little.

When the first edition of this volume was written the physiologists, and especially Pawlow and his pupils, were revealing to us many very interesting truths concerning the relations of the stomach and the nervous system; as, for instance, that severance of the vagi arrests the secretion of the stomach or renders it abnormal; that irritation of these nerves excites the secretion; that food cannot enter the stomach, or indeed the mouth, or even come within the fields of sight or smell without provoking a lively secretion of gastric juice; that emotions of grief or anger may arrest the secretion; that the absorption of certain products derived from the portion of food first digested heightens the secretory activity of the gastric cells; and so forth. He has shewn us how in the dog even the sight of meat promotes a flow of gastric juice, but after an interval of five or six minutes, a period of latency which seems to be occupied by intermediate glandular processes. In thirty-five minutes the secretion will amount to 17·8 c.cm. Now, if the dog be tantalised with meat until his desire is exhausted, the secretion of gastric juice does not occur, or is arrested. These observations throw a strong light on the common experience of digestion in man. If with certain precautions the vagi are divided in such a dog, the secretion of gastric juice ceases, in three or four days dyspepsia becomes manifest; food lies in the stomach and putrefies, and the dog wastes and dies of the poison. But if, twenty-four hours after the division of the vagi, one of them is withdrawn from the wound and its distal end stimulated, gastric juice, after the usual interval of latency, is secreted again.

Concerning the sensations of the stomach it seems that we know little. The ingestion of cordials, or of warm liquids, is evidence of one kind of sensation; the symptoms of indigestion or of ulcer suffice to prove the sense of pain, though it is not clear how far gastric pain is to be attributed to drag upon the peritoneum during peristalsis or to irritation of this investment. Now pain, whatever its machinery, may arrest or modify gastric secretion; and hyperaesthesia of the stomach, like emotions of the mind, may either inhibit the formation of gastric juice, or excite hyperchlorhydria. On the other hand, torpor of smell or taste, and possibly an anaesthesia of the stomach, may arrest or pervert the secretion which should follow the ingestion of food; the food may not be recognised,

as it were. Thus, monotonous or insipid diet, absence of mind in care or excitement, or factitious indifferences to food may deprive the stomach of the due quantity or quality of gastric juice. We often observe that articles of diet of less digestibility may agree better with one who enjoys them than other diets, presumably better, which are unwelcome. Thus sometimes "cravings" may well be indulged. That a careless dinner eaten with relish and mirth is often better digested than a dinner dictated by the physician but eaten in the solitude of the chamber, is well known. If depressing emotions diminish the values of the gastric juice, exhilaration probably increases them.

Again, as certain nervous influences govern secretion directly, so indirectly also other nerves control the vascular supply to the organ; but of the precise concern of these in digestion, and of their relation to the direct nerves of secretion, we know little or nothing except that activity of the mucous membrane is associated with reddening of its surface. Whether in any disease the nerves of the blood-vessels are primarily affected, and so consequently the functions of the stomach, we do not know. We do know, however, that the stomach, like the heart, has to provide for its own nutrition; and, like the heart, often falls into the vicious circle of doing as ill for itself as for other parts of the body. Whether the vasomotor or other nerves protect or nurse the stomach as the vagus nurses the heart, we do not know. But it is clear that, like the heart, the stomach is possessed of a certain tone; and that this tone is not constant, but varies largely with circumstances and capacities. Now tone must be largely under the influence of the nervous system; but, on the other hand, the muscular quality is often reduced by bad or deficient blood, whether the blood be simply poor or charged with poison which may be brewed in the stomach to its own injury, as, for instance, in dilatation of the cavity; or, as in the infective fevers, it may have other modes of origin. This argument does not take us altogether away from the nervous system; for probably many of these poisons, such as choline, act through this system. A stomach by nervous defect may lose tone, food may be delayed in the cavity, poisons may be brewed there, and these may not only taint the blood, but may also so impair the nervous parts themselves that the stomach may suffer both primarily and secondarily from the nervous system. In certain cases of neurasthenia, or pseudo-neurasthenia, the stomach is the primary source of the departure from health; in others, if indeed the initiation of the morbid series lay with the nervous system, the secondary impairment of the stomach may have become the central feature, and the alleged neurasthenia be due to bad products of digestion which, returning again to the stomach, depress it and its work still further: if so, the direct way to a cure is by way of gastric juice medication; in the vicious circle the stomach is the link which has to be forged anew. Certain poisons, such as tea, coffee, and tobacco, seem to have an immediate action upon the gastric nerves; and the list might be much extended. Some of these agents are discussed at length in other sections of this *System*.

Furthermore, in the phenomena of particular diseases we find evidence of the direct dependence of the stomach upon the nervous system: in asthma we have a malady which often concerns the stomach almost as much as the lung; the vomiting of cerebral disease is to be observed in every hospital; the vexed and windy stomach of chronic heart disease seems to consist in something more than the venous congestion of it, as its unrest is not confined to cases of high venous pressures. Seeing, then, that the nervous part of the functions of the stomach, whether normal or abnormal, is perhaps but another expression for the dynamic aspect of these functions—for at this moment let us disregard the sphere of consciousness in the matter—it is evident that the nervous element in the causation of gastric derangements is all-pervading; while on the other hand it is no less evident that to consider the neurotic factor alone would vitiate every argument on the subject. If, then, nervous disorder finds its expression in terms of gastric juice, or in a relaxation of muscular tone, should we not do better to omit this chapter and to dwell on these local changes, in terms of tone and secretion as they are more positively manifested in the affected organ itself? Would it not be better to deal more positively with the nearer than more conjecturally with the remoter links in the chain? The reply of the working physician is that these nearer changes are less positive than they appear: to work efficiently we must know the remoter as well as the proximate disorders, the initiations as well as the issues of change; and, indeed, the dynamical oscillations may sometimes be even easier to recognise, if not to measure, than the more statical—that is, of course, until we enter upon the study of the grosser forms of disease. For instance, a nervous disorder may and probably will affect other organs as well as the stomach; if the disturbance begin in the nervous system, gusts of it will probably be observed in various regions, although perhaps in none can the results be directly perceived or measured. Again, if tone in its largest acceptance be something more than the mere preservation of the capacity of a hollow organ about its own normal mean, and signify the preservation of that degree of mutual tension or polarity between all organs which, like that existing between the several members of the solar system, keeps all parts in balance and in reciprocal service, in this view of the subject a relaxation of tone might be more readily perceptible in the rocking of the whole system, or in a slackening of tension between particular parts, than in certain variations of secretion and the like within the organs themselves, which might be hard to appreciate. Thus, for example, the practical physician will say that a nervous dyspeptic is easy enough of recognition: in addition to the disorders of the stomach he presents certain general characters which stamp him as a "neurotic," as one in whom nervous capacity is diminished, or nervous quality perverted. Not only so, but with a little care we may pick out in a rough way the cases in which the stomach seems itself to have been the origin of the malady from those in which the stomach has suffered secondarily or, at any rate, in association with other parts—such as the uterus or the colon. If the physician, by virtue of his

knowledge of the world, recognises his patient as one in whom nervous intensity is evidently greater than physical capacity; and if such an one complain of neuralgia of the head and face, of fits of exhaustion, of sleeplessness, of irritability, of loss of self-control in little things, of palpitations perhaps, and of muscular restlessness; if he be lean and bloodless, if his demeanour be excitable and impulsive, and if withal he complain of dyspepsia, it is perhaps true to surmise that the nervous system is primarily at fault and first needs the physician's attention; although the patient may complain most loudly of his stomach and, with the curiosity of such persons, may inquire for bismuth, soda, gentian, arsenic, and like "cures for indigestion." Or, again, an anaemic young woman, rather lethargic than vivacious, may come to the physician complaining of severe pain in or about the stomach, or, on the contrary, of utter aversion to food, or of vomiting; and if besides she has fits of polyuria, weakness or numbness of limbs, tenderness of certain points on the spine, contracted fields of vision, and the like, the physician must decide whether her distress be due primarily to some gastric lesion or to hysteria, a problem which can only be solved by a consideration of all the features of the person herself, as well as of the manner of the pain: to make the diagnosis by restricting ourselves to irregular chemical tests of the stomach itself and its secretions would be more easy and more fallacious. A third patient may present himself with gastric pain and with vomiting, but also with a recent squint, with pains in his legs and a disorder of gait; is such a case one of "nervous dyspepsia"? Not to multiply instances, these are enough to shew that even in the majority of the disorders of the stomach the nervous element may be largely concerned, and that there is a proper place for the clinical study of many stomach disorders from a nervous point of view. The difference may be one of degree or one of origin; and the difficulty of writing this article lies in the danger of deviation on the one hand into primary dyspepsias, on the other into neurasthenia, hysteria, tabes and other diseases of the nervous system dealt with in Vols. VII. and VIII.

Causes.—It is chiefly by their causation that the neuroses of the stomach are known. Take a simple instance: let us suppose that a vigorous young man of twenty-two goes out to Switzerland, and engages in a first-class excursion before he has got into training; he comes home to his inn after eighteen hours on foot and falls upon a large supper, which he probably washes down with new Swiss champagne. This done he rolls off to bed heavy and weary, but his sleep is brief: in the small hours of the morning he becomes aware that he is restless, burdened at the stomach, and sickly. If he had vomited he had been lucky; or wise if he had taken an emetic: but he passes a restless night, feels "bilious" all next day, and is out of sorts for more than a day. A smart attack of diarrhoea is no uncommon consequence, but a costly remedy, and one which might leave him out of condition for a week. If vomiting does occur the food comes up half fermented, but much as it was eaten, for digestion had scarcely begun. Another time he will know better; he will take a

warm bath and turn into bed with no more than a basin of soup and a thimbleful of brandy inside him, and he will eat his supper next morning. Now what is the meaning of such dyspepsia as this? That the stomach failed because the nervous energy was exhausted (*vide* p. 367).

The explanation is the same in the case of a keen man of business who takes no rest; he drives himself and those about him at full speed: every disappointment, every mishap worries him and he becomes nervous, fretful, sleepless, and—dyspeptic.

Or a little boy or girl is born of anxious, nervous parents; his life is artificial, he walks in his sleep, he is very excitable at play, and after play is tired out; his lessons make his head ache; if he catches some infectious ailment he starts and twitches or has even a convulsion. Now if we examine his body we shall see that his legs are thin, but his belly tumid and squelchy on palpation; his bowels are constipated; he takes little or no breakfast, and he is qualmish and whimsical at his meals. Between meals he may take tempting morsels, but not enough to supply the wants of active growth. Moreover, now and again he has inexplicable febrile attacks with vomiting, or lienteric diarrhoea, which no ordinary rule seems wholly to prevent. In these cases, again, the nervous system is ill controlled, or wanting in capacity, or both.

Once more, gastric disturbances, apparently due to the mediation of the nervous system, are wont to arise in sympathy with disturbances elsewhere; as, for example, in the uterus, in the ureter, in the bile-duct. These sympathetic gastric neuroses are well known, and I may be content with an indication of this side of their pathology.

To say, then, that "nervous dyspepsia" is particularly a disease of middle-aged and harassed men and women is to erect a group of symptoms into a "type," and thus to hold them aloof from other groups of symptoms with which, perhaps without much superficial resemblance, they have nevertheless an intimate affinity. We cannot, therefore, construct a pattern of the disease, but the symptoms must be dealt with severally, the relations of each being indicated as far as possible.

Symptoms.—*Pain and other Sensory Perversions.*—"Every schoolboy knows" how the stomach can ache; and, on the other hand, the physician is too often witness of the pain of ulcer or malignant disease of the organ. In the aching of a stomach or bowel which resents an unwholesome morsel there is nothing abnormal; but in some persons the stomach aches badly however bland its contents, nay, when it is altogether empty. Such pain is called gastralgia, and it is often a matter of no little difficulty to distinguish this pain from the pain of simple ulcer or of cancer. A pure neuralgia, apart from temporary irritants, is not common before adult years; but it may continue thenceforward with intervals of relief up to old age. In the gastralgia of old persons, in whose case a suspicion of malignant disease more easily finds its way into the mind of the physician, we note, perhaps invariably, a previous history of the former malady, or of others akin to it, which, although not contradicting the supposition of graver disease, may suggest a more hopeful opinion. In

character the pain varies; in many cases it is a wearing or gnawing pain, very hard to bear; in others it is a sinking rather, or a "misery"; in others it is acute or lancinating, when the pain may radiate into the abdomen, into the loins, or into the thoracic, cardiac, and intercostal regions. The pain of mere neuralgia—of neuralgia, that is, apart from local disorder of the stomach—does not usually penetrate to the interscapular region: I am far from saying that it never does, for I have met with it occasionally, but such a direction of pain increases the suspicion of ulcer. In neuralgia the pain or tenderness, more frequently lower down the back — about the 9th thoracic vertebra — is often relieved by pressure, even by strong pressure, as by leaning over the back of a chair; and though in not a few cases there is some superficial tenderness, yet this is more diffused than in ulcer, the tender area of which is often so small as to be covered by the point of the finger; the tender area of gastralgia is usually at least as large as the hand, and the skin is often hyperaesthetic over a much larger surface. Points of definite tenderness, and areas of hyperaesthesia, have been mapped out for gastralgia as for other neuralgias and for ulcer; hitherto, however, I have found them too inconstant and subjective to determine a doubtful diagnosis. I see Mr. Mansell Moullin writes that cutaneous hyperaesthesia may occur without ulcer. Féré is of the same opinion. Mr. Moullin adds, however, that persistent deep tenderness with cutaneous hyperaesthesia is strongly suggestive of ulcer.

If the attacks of pain occur independently of food, as for instance in the middle of the night, or before breakfast—no uncommon hour for its recurrence—the diagnosis is easier than when the pain recurs, as not infrequently it does, immediately or at some regular interval after meals. Systematic pain, constant in seat, constant in periodic recurrence, is at the least highly suggestive of ulcer, and vomiting is by no means a constant in ulceration. The sufferer under static disease may well be agitated and distressed, but the nervous dyspeptic with his "little papers," his caprices, and his self-absorption usually makes a different figure. The one will win your sympathies and your help, and will keep them, the other goes near to become a bore. Even when pain occurs soon after meals, however, vomiting is usually, though by no means always, absent in cases of neuralgia; on the contrary, pain and vomiting recurring with some regularity after food may be proved, even by surgery itself, to be of "functional" origin (*vide* p. 479). Nay, we cannot positively say that pain, vomiting, and even haematemesis are decisive against gastralgia: some women have a way of vomiting blood, even for years, without recognisable ulcer. I recall not a few cases in which this triad of symptoms meant, after all, no more than "functional" disease of the organ. Without post-mortem examination, of course, a positive diagnosis is impossible; yet in particular cases there are circumstances, such as hysteria, to create a strong opinion in favour of the lighter diagnosis. One remarkable character of these eccentric cases is that the patient, instead of being blanched and reduced for many weeks or months, soon recovers a fair complexion and

bodily condition, if indeed she ever loses it. The pain of over-acidity of the stomach and that of gastralgia may be much alike, but the former is more entirely relieved by a sufficient dose of an alkali, or by nitrogenous food. Gastralgia is also usually not constantly relieved by food; but there is a certain troublesome kind of gastralgia in which the ingestion of nourishment, even the blandest, may be followed by torments. This kind of attack is not infrequent in neurotic young women, and in them the dread of food becomes so besetting that the sufferer cuts it down to the lowest point, and may thus reduce herself to extreme emaciation. In these persons, however, the pain is not quite like that of ulcer; it is more unreasonable; in ulcer to swallow a spoonful of gruel is not instant torture: still there are milder cases of this kind of hyperaesthetic gastralgia in which the differential diagnosis cannot be made by the pain taken alone, though the other features of the case usually suffice to determine a right conclusion. At the same time it must be remembered that in some cases the pain of an ulcer is associated with pains which are rather of the nature of gastralgia, as if the local disease awakened a gastralgia; and the physician will do well to bear in mind that pains having the characters of gastralgia do not exclude ulcer, the direct pain of which may indeed be less importunate, or more or less merged in the neuralgia. The pains of malignant disease are usually aggravated by peristalsis; but, whatever their behaviour, gastric pains awakened for the first time in a person at or beyond middle life must give rise to anxious suspicions. It is said, I do not know with how much truth, that the pain of an ulcerated surface is also aggravated by a dose of alcohol or of strong salt and water, while the pain of gastralgia may be relieved. Again, the ordinary restrictions of diet, which usually relieve other disorders of the stomach, do not relieve gastralgia; for the digestive powers of gastralgic patients (as ascertained by test meals) are really not much deteriorated. It is rarely wise to use the siphon frequently in gastralgic cases, but in a few of them there is evidence of delayed digestion, and of a tendency to variation in the secretion of hydrochloric acid—generally in the direction of insufficiency, but sometimes in that of excess. In my recent experience, however, estimates of free hydrochloric acid, estimates made by experts in the test, have proved very inconsistent, not only as between case and case, but also during the course of individual cases. Probably hyperchlorhydria and gastralgia are closely allied, as I have suggested, in some tobacco smokers, a suspicion in which Dr. Norman Dalton has expressed his agreement.

Pain, again, may be left as a legacy by foregone disease, as by a gastritis. From some closely observed cases of ulcer I have surmised that recurrences of pain are not always dependent upon a return of the ulceration itself; but the pain by its characters and lines of reflexion betrays itself as an echo of the distress of the primary disease. Such cases, however, end in complete recovery, unless the pain be due to an adhesion. The disposition of gastralgia to increase about the menstrual period may be a point of difference between it and ulcer. Another

difficulty may lie in the distinction between gastralgia and angina pectoris. Epigastric angina is not very rare, but in the true disease the circumstances of the individual case will surely guide the wary observer. Between the so-called "false anginas" and gastralgia the distinction may be difficult, though not of much practical importance. The proximate causes, the agitation, the gaspings, and the paroxysmal modes of recurrence will lead to a right interpretation of attacks which generally consist in severe intercostal neuralgia with vasomotor storms; thus, conversely, abdominal cramps, abdominal pulse, and other features may incline our opinion to gastralgia; but the two may well overlap, or may be associated with other neuralgias. To add to our perplexity a remarkable alliance, even in non-tabetic cases, between what seems to be a gastralgia and aortic regurgitation has been pointed out by several writers, by myself among others. No definite explanation of the coincidence has been proposed (*vide* art. "Aortic Disease of the Heart," Vol. VI.).

The gastric crises of tabes dorsalis must be diagnosed on their own evidence; in itself the pain may not be decisive, but its violent and paroxysmal character with intervals of total absence are unlike gastralgia, which, if not continuous, is more importunate. The degree of pain in gastralgia may, however, be quite as great as in tabes; indeed, it is sometimes so violent, both in itself and in the cramps which attend it, as to throw the sufferer into a collapse. I believe I once saw such a collapse prove fatal in a highly neurotic woman, long a victim to abdominal and other neuralgias; her pulse failed, her limbs and face grew cold, and she could not be brought round: the absence of a necropsy precludes me from speaking confidently, but I once saw such a death follow the passage of a gall-stone in a healthy young woman; in this case an autopsy was made, and with the exception of the stone, which was found in the uninjured common duct near its entrance into the gut, the body was perfectly healthy. We must not assume, therefore, that mere abdominal pain cannot be fatal.

On the other hand, the pain of gastralgia may be very slight; often it amounts to no more than a sense of weight or fulness in the pit of the stomach, or a restless uneasiness or sinking, more teasing than violent. A sense of heat rather than pain is sometimes the complaint of nervous dyspeptics. One patient—a middle-aged serious man—described the heat to me as burning all down his breast-bone and epigastrium, and even into the abdomen; it was like "liquid fire," he said; and it was made worse by food even of the simplest kind. This symptom, not uncommon in more moderate degrees, I regarded as due to hyperaesthesia, and not to Prout's hyperchlorhydria. There was no evidence of over-acidity, nor was the burning relieved by large experimental doses of alkali. He told me that light sponge-cakes settled best with him; and he could take eggs in weak tea without aggravation of his sufferings.

Gastric distress in neurotic persons, then, is not always pain properly so-called; often it consists in perverted organic sensations which may,

however, be no less hard to bear. Of these perhaps the commonest is the sense of sinking, which seems to be peculiarly distressing; this distress is not uncommon in intelligent and self-controlled persons. Such an one describes the sensation, referred to the epigastrium and upper abdomen, as though of utter exhaustion; the voice becomes weak and slow, and the face turns grey; but the attack does not feel like a faint, nor lead to it. Sometimes this sensation is due to the use of tea by persons intolerant of it; but it is common enough independently of such agents. Food does not relieve it much, and happily alcohol is no very rapid means of comfort. A cup of coffee or of hot beef tea well spiced answers perhaps best, and is at least harmless; patients who suffer thus are liable also to false hunger, an acute and depressing want rather than a normal appetite; food soon brings satiety, but not much relief. Mid forenoon and late afternoon are the usual times for this discomfort; or it may come on in the middle of the night. In many persons a saline purgative is sure to bring it on. It is due, in my opinion, to splanchnic vascular dilatation, for the radial pulse usually falls in pressure; and a more definite criterion is to be found in the excessive difference in pulse-rate between that of standing and lying down—as, for instance, between the numbers of 70 in recumbency and 110-120 on returning to the upright position, an acceleration which does not abate much until the patient lies down again, when, after a few variable and intermediate pulses, the rate falls to normal almost suddenly. These phenomena I have found a very valuable aid in interpreting "sinkings" and "exhaustions"; in various degrees they are frequently obtainable, and are by no means confined, of course, to gastralgic cases.

Bulimia is a rarer phenomenon, and I am not sure that this perversion is to be considered with gastralgia, but it seems to occur in neurotic subjects. Some of these cases are of astounding enormity. One I remember of a young man of some thirty years of age, in whom the craving was prodigious. Not only did the patient eat ravenously during the day, but he could not pass the night without gorging himself more and more. His landlady, a kindly soul who grudged him nothing, was unable to repress a tear when she told us that he would call for as many as thirty huge sandwiches between night and morning, sandwiches two to the slice of the quartern loaf, of which he would consume ten well lined with ham or beef, and often would ring once or twice for another plateful before morning. The poor woman had contracted to board the patient at a price; now she bore a divided mind between anxiety lest she should pine her pensioner and lest by him she should be brought to bankruptcy. In less degrees, scarcely to be called bulimia, this craving is not uncommon; and it is alleged by some observers that it depends upon an affection, temporary or organic, of the pneumogastric nerve. I have seen it notably in Graves' disease. In one recorded case of it a neuroma was discovered upon one pneumogastric trunk.

Vomiting.—Ever since as a novice I read Watson's *Lectures* I have been on the watch for cases of the malady clearly described by many

writers, and very vividly by Watson, under the name of pyrosis. Morning vomiting or puking is common enough, especially in topers; so it is in those afflicted with renal and other diseases, and vomiting is far from uncommon in neurotics who take no excess of food or drink. But this is not pyrosis, which must have become more rare, for the description was obviously taken from nature. I used to ask out-patients over and over again to describe their morning qualms or vomiting, and in private practice I have inquired after similar symptoms, but of specimens of the classical pyrosis I secured very few; race, or habits, or district, or luck seem to have been against me.[1] The vomiting of gastralgia may occur at any time of the twenty-four hours; for the most part it is excited by food, which in this case must be reduced in quantity. Such vomiting occurs chiefly, but by no means exclusively, in women, and these women young; it may also occur in males, but always, I think, in young adults or children. Dr. Sidney Martin gives a case of the kind in a young woman who vomited every meal for intermittent periods during three years. The curious thing is that these patients often keep fairly plump in spite of their waste of good food; no doubt some considerable part of it is retained. Dr. Martin says that these patients are always neurasthenic or hysterical; yet I have seen many cases which could not thus be classed, unless we class thus all persons of keen temperament, or in whom there is a morbid reflex irritability of any organ, with or without other symptoms of the more general malady. The cases are very intractable, especially if treated at home, when such patients, if in comfortable circumstances, may soon be nursed into hysteria: but unfortunately the affection "functional vomiting" is not confined to the well-to-do, and breaks out also in poor, hard-working women, who are reduced to great straits by it. In them probably the causes are over-work and ill sustenance, such as a spare and monotonous diet of bread and tea. Watson describes a case after scarlet fever in a girl of sixteen; she was cured by restricting the diet, not to frequent small quantities, but to one meal a day. He truly points out that in these cases nausea is infrequent. Some dilatation of the stomach may be present, but, when present, it is a subordinate and transient feature, as I know from the use of the siphon in some of them.

Regurgitation and even rumination of the upcast food are curious features of some cases of nervous irritable stomachs (Merycism). The trick, for such we may almost call it, is not necessarily associated either with pain or vomiting, but apparently with some periodical or persistent patency of the cardiac sphincter. In extreme and rebellious degrees it is common in chronic insanity, being most frequent in imbeciles, with many of whom it is a lifelong habit; cases are, however, recorded in normal and even intelligent persons (2A).

Flatulence, which may or may not be associated with pain, is a remarkable "neurosis" of the stomach. It generally rises in storms, with such volumes of noise and gust that one wonders whence it can

[1] Sir Lauder Brunton seems to explain the ambiguity, p. 354.

come. Curiously enough distension of the epigastric region stands in no direct relation to the flatulence; the two events may concur, but the sense of distension, which may be so great as to oblige the sufferer to undress, may not be associated with measurable enlargement of the waist; yet the wind may be tremendous nevertheless. To pursue this matter far would lead us into the subject of hysteria, especially as regards wind swallowed as such and regurgitated. In these cases, as Graves pointed out, there seems to be some source of flatus other than decomposition of food. That air may be poured into the alimentary canal from its own walls is possible, one can neither aver nor deny it; and, if this be so, it may explain some of these strange phenomena. A sensible, hard-working professional man may tell us that not unusually he wakes in the small hours of the morning, sits up in bed, and rolls up wind, belching it forth boisterously for many minutes. After some repetitions of these performances he lies down to sleep again. I need scarcely say that such an one is a light sleeper at the best; that he is a spare, sallow, restless man; and a nervous dyspeptic of the classical kind. But a young woman apparently sensible, and of a calm and equable disposition, came to me not long ago in fear of losing a good position as mistress of an elementary school, on account of these volcanic rumblings and noisy upheavals of air, which of course "made her impossible." An attack came on in my room, a convulsion of eructations; but before we could get her corsets off it ceased, and the physical signs were negative. Her pulse was unaffected. She solemnly assured me that she never consciously swallowed air, and was willing to make any sacrifice for a cure. I fear I did her no good. With the flatulence disturbance of the heart may be associated; but to follow this symptom beyond its mechanical causes would lead us into a general discussion of hysteria and neurasthenia. Dr. Goodhart states that the neurotic flatulence of man is more commonly in the stomach, of woman in the bowels. The vented gases are, as a rule, inoffensive; if offensive, there is more than gastric neurosis in the case; though I ought perhaps here to except those obscure cases in which *sulphuretted hydrogen* is discharged from the stomach, often in such quantity as to make the whole atmosphere about the patient offensive. The only instance of this kind in my experience was in a young woman, undoubtedly neurotic, and neurotic only (*vide* art. "Dilatation of the Stomach," p. 535).

Anorexia Nervosa.—Under the convenient title of anaesthesia I have referred to a state of stomach in which the natural sensation of hunger is blunted or even absent. Such cases are met with, always perhaps in neurotic women, but not necessarily associated with other symptoms of hysteria; of neurasthenia I will say nothing, for this malady cannot persist without weakness of all the functions of the body. To these not uncommon cases, already described by other observers including myself, Sir William Gull gave the name of Anorexia nervosa, and the name is a good one. When for many months food has been taken in utterly inadequate quantities, neurasthenia (in its etymological sense) must follow, as likewise asthenia of all and any other systems of the emaciated

body ; yet it is a remarkable character of these patients that they continue capable of occupations, interests, and even of efforts which, if not in themselves extraordinary, are at least astonishing in such frailty. A young woman thus afflicted, her clothes scarcely hanging together on her anatomy, her pulse slow and slack, her temperature two degrees below the normal mean, her bowels closed, her hair like that of a corpse dry and lustreless, her face and limbs ashy and cold, her hollow eyes the only vivid thing about her—this wan creature, whose daily food might lie on a crown piece, will be busy with mothers' meetings, with little sisters' frocks, with university extension, and with what you please else of unselfish effort, yet on what funds God only knows. At meal times her mother may cry, her father may storm, her friends may banter, and the cheerful reply never fails, that she has eaten amply ; or, if not, that she can eat no more. Every physician of experience will say that this is no overcharged picture. Many young women indeed, of ardent and self-forgetful nature, and happily there are many such, go near to fall into this state. Eating seems to them a crass matter ; meat, even the smell of it, makes them sick ; they declare that they eat as much as other people : nay, pathetically enough, these very ascetics are often beset by the notion that some other member of the family circle is not eating enough. Gradually the appetite neglected begins to disappear, perhaps for want of elements in the gastric secretions which are dependent on demand ; and so by degrees, and without the patient's own consciousness, the body is painlessly pined. It has been stated that a superficial cutaneous area of anaesthesia, corresponding to the area of the stomach, is characteristic of anorexia. My own clinical experience does not bear this out, nor, I believe, the researches of Dr. Henry Head. Such anaesthetic patches probably form but a part of the distribution of hysterical anaesthesia. As I have hinted already, the senses of taste and smell may prove to be abated. I have never noted dilatation of the stomach in anorexia nervosa ; in extreme cases it may appear, perhaps, but the stomach is usually small or retracted. In minor degrees this state of appetite may be recognised more or less in men also ; and at all ages short of old age. In old persons such a failure is of a different nature, and means at least an atrophy, whether of the stomach itself or of other parts, which is no mere neurosis. With anorexia nervosa gastralgia, or hyperaesthesia of the stomach, may or may not be associated ; cases of the one are often seen without the other. Vomiting, which seems to belong rather to another chain of gastric neuroses, or of course to gastritis, is usually absent. It is not unlikely that a numbness of the stomach in the hypochondriacal or melancholic gives rise to the delusion of vanished stomach.

Motor Disorders.—Of symptoms concerning the motor functions of the stomach in gastralgia there is not much to say. It is notable that atonic distension of the stomach is not very frequent in gastralgia, but to loss of muscular inhibition may be attributed the manifold gurglings and other noises to be heard only too often in such persons, often without pain,

and save by the ear unperceived by them. In society these murmurings, and other audible noises of the gastric machinery, are often very untimely, and our aid may be sought in the removal of them. This is no easy task; they can be dealt with only on general principles. It is too often alleged that in such stomachs the organ or its muscle is in such an irritable fuss that the food is hurried with undue rapidity into the intestines, and proper digestion thus prevented. This may be so; the stools of such patients must be inspected in order to see whether the food passes through imperfectly digested. However, so far as I have made any investigations which may deserve the character of scientific, delay rather than acceleration was discovered; the motor disorder is usually, I believe, in the direction of defect rather than excess, though no doubt, if excited by abnormal contents, the gastric muscle has times of resentment. In these fretful stomachs a false splash, a splash which might be taken for the splash of dilatation, is often to be obtained; but to the expert physician the sounds are not closely alike; that of gastric atony is a squelch, that of dilatation is a slop, and in a baggy stomach the toss of the gastric contents is often much more palpable. It is not unlikely that during cramping pain the stomach or the pylorus is spasmodically contracted; indeed on inspection the epigastrium is often retracted, and the recti abdominis may be tight.

On the influence that nervous irritation or arrest may have upon the *secretions* of the stomach I referred to Pawlow's researches.

When we turn to consider *symptoms other than gastric* which are associated with gastralgia and its allies we are in danger, as I have already said, of travelling out of the field, and of trespassing upon that of neurasthenia or of hysteria, of hypochondriasis or of melancholia, maladies with which disturbances of the stomach are often associated, whether as cause, consequence, or coincidence. Thus, symptoms of what is called a "predisposing" kind must be omitted in this place. But certain symptoms which may be regarded as subsidiary to or even consequent upon gastric neuroses, must be briefly enumerated. In severe gastralgia the pulse may be much retarded and lowered in pressure, the face and limbs may grow cold, even the teeth may chatter. In the lower part of the alimentary canal colics, windy tumidities, and occasionally phantom tumours arise, and therewith light diarrhoeas are often associated. In the bowels, moreover, the gastric drama is often re-enacted. In the bowels pain as severe as that of the stomach may arise, with borborygmi and hyperaesthesia of the surface which, together with the tenseness of the abdominal walls, may arouse suspicions of inflammation. Trousseau says with truth that colalgia may be so closely associated with gastralgia as to be indistinguishable from it. In March 1904 I read to the Cambridge Medical Society a paper on Spasm of the Colon, in which I described certain observations which were more than supported by Dr. Hawkins in 1906. We have pointed out that in these cases, mostly but not always in women, the colon—usually the caecal and ascending portion—constricts itself, and may be felt like a sausage or a snake. In the

right iliac fossa appendicitis may thus be simulated, and so closely that twice I have seen the parts opened for removal of an appendix which in both proved to be quite healthy. In emaciated persons the bowel may be seen plainly; in a case Professor Saundby kindly asked me to see, this contortion, which partially or sinuously affected the whole colon, was as visible as it was palpable. The pain was intense.

Of symptoms still further afield may be mentioned polyuria—which does not necessarily signify hysteria, vertigo, panting, palpitation, throbbing of the abdominal aorta, and yawning sluggishness after meals. To say that these symptoms, or some of them, prove the existence of hysteria is to assert that all neurotic perversions are of the nature of hysteria.

Finally, many gastralgics are liable also to asthma, and again to eczema. These affinities are often to be recognised in the course of private practice, where careful evidence of past illnesses and of pathological pedigrees can be obtained.

Diagnosis.—In the course of the analysis of symptoms we have incidentally considered certain points of diagnosis, for instance the distinction between gastralgia and ulcer, and to these I need not return. If ulcer be set aside the greatest difficulty will be found in distinguishing between gastralgia and gall-stones. At a given moment the differential diagnosis between neuralgia and gall-stone may be impossible, and, at any time, it must often be inferred from the history of the case; I am contemplating, of course, those cases of gall-stone colic only in which jaundice is absent—cases far more numerous than is generally supposed. We must not be ashamed to admit that it is often impossible to decide at once between the one disorder and the other; still, in most cases the occasional and comparatively rare occurrence of seizures in the midst of apparent health will enable us to make a true diagnosis. The difficult cases are those in which the gall-stone pain becomes almost chronic, yet without jaundice, and in which, by the repetition of attacks, the patient is unnerved. I need not say that gall-stones may properly be suspected in young and clear-skinned persons as well as in the middle-aged. By the immediate characters of the pain I think that no sure diagnosis is to be made, at any rate not always. In gastralgia shivering, even to chattering of the teeth, without elevation of temperature, is not uncommon. Another serious difficulty in diagnosis lies between neuroses, gastralgia especially, and malignant disease of the stomach; such problems arise in patients at or after fifty years of age. Gastralgia is by no means unknown in persons advanced in life, and indeed in men and women, not perhaps highly neurotic, and certainly not hysterical or melancholic, but of anxious or irritable habit, who may be reduced in health by overwork or harass, and have fallen into that state of indefinite ailment which so often appears and departs during the time of transition from maturity to age. In these circumstances gastralgia or unaccountable vomiting may appear for the first time, and without previous history of manifest neuroses. In them, as in possible subjects of ulcer, if the pain be systematic in its recurrence, if it is almost inevitable at a certain earlier or later stage of

digestion local lesion is probable; if it occurs when the digestive process is well past it may be hyperchlorhydria or gastralgia—maladies closely akin, often perhaps identical. Now if a man or woman of fifty-five to sixty-five years of age, losing the bloom of maturity and falling somewhat in flesh and strength and appetite, consult a physician for pain in the region of the stomach, diagnosis may be very difficult, and on one visit impossible. The patient must be put to bed, closely watched, and very carefully examined on every visit; a thickening of inner parts may on one occasion be imperceptible yet perceptible on another. If, thus watched and carefully treated, the patient gain some weight, malignant disease may nevertheless be present, and no assurance of safety may be possible until a month or six weeks of supervision have elapsed; for we must not forget that pains of gastralgic character, and even of gastralgic nature, may arise under the irritation of local disease. In these dilemmas examination of the faeces, in which I have been ably assisted by Dr. Cammidge, has been far more valuable in my experience than stomach probings, and has often given me crucial facts. The two most useful observations have been on defective assimilation of fat and on occult blood [*vide* p. 466]. Fat digestion scarcely falls within the present subject, but the presence or absence of occult blood in cases of doubtful interpretation proves over and over again to be always highly significant and often decisive. On the other hand, stomach testing, in more than one case of grave apprehension in tired, worried elderly persons, has dictated, on deficiencies of hydrochloric acid, diagnosis of malignant disease, where complete recovery has followed, or where on death from other causes an autopsy in respect of malignant or ulcerative disease has proved negative. An instance of gastralgia due to the dragging of an old adhesion is given in Vol. II. Part I. p. 951, and the reader is referred also to the article on "Visceroptosis" in this volume. Between gastro-enteralgia and peritonitis a doubt may arise. Great superficial tenderness and severe inward pain, together with a nipped expression of face and small pulse, may combine to the physician's embarrassment. The thermometer is of little use here. The history of the patient and of the attack will scarcely fail to give some help in diagnosis, and unimpeded action of the diaphragm would be a favourable sign, if, for example, perforating ulcer were suspected. Hiccup, vomiting, and meteorism, if present in any degree, will scarcely simulate those of peritonitis. In a word, no great difficulty is likely to occur. A hint first given to me many years ago by the late Mr. Jessop of Leeds, I have found invaluable; namely, to scrutinise the navel for the tiniest hernia of bowel or omentum. This hint has put me in the way to interpret and to cure many a case of obscure recurrent "abdominal neuralgia."

Finally, in all cases of pain about the waist and abdomen the knee-jerks must be tested, and the vertebral column closely examined. If the knee-jerks be absent, the diagnosis of neuralgia in its general sense must at any rate be postponed.

Treatment.—The treatment of gastric neuroses is of two kinds or

purposes. In bad cases immediate relief is urgently needed; in all cases general treatment is needed to cure both local distress and the state of system to which the malady is due. First let us suppose that the gastralgia appears in a young woman whose general condition is not one to cause much anxiety. She may be anaemic, and iron may bring relief; or if not, a few drops of Fowler's solution will pretty surely secure this end. It is needless to say that the diet and mode of life must be carefully ordered. Next let us suppose the case to be in an older person, of either sex, of neurotic habit, and reduced in flesh and strength by pain, refusal of food, or adverse circumstances. The first thing to be done is to put the patient to bed for a week or more; no excuses arising from mere restlessness are to be admitted. Many a bad case of gastralgia, as of other neuralgia, has been cured by a fortnight or three weeks in bed with careful management of diet, perhaps with restriction to milk for a few days; the warmth, rest, and seclusion from affairs, the coaxing of bland food into the weary stomach, and the administration of hydrocyanic acid, or cherry-laurel water, and bismuth, being the chief agents of relief. If skilful massage can be added, light and brief at first and increased as the strength will bear it, the recovery will be facilitated. But, as I have said, it is undesirable to focus the mind of the gastralgic subject on his malady, as by lavage, or even by much testing, proceedings which are apt to create or to develop what Déjerine calls "les fausses gastropathes." If after the first two days in bed the pain continues severely, let opium be given. It is better for obvious reasons to avoid the hypodermic syringe; and, fortunately, in these cases no means answer better than small doses of the solid drug administered in a pill, of the contents of which the patient is better ignorant. I have found Dover's powder in doses of three or five grains, with an added half-grain of ipecacuanha, very suitable; small pills or cachets are often retained when liquids are vomited, and I think the gradual solution of the pill is an advantage. In this pill, or otherwise, proper means may be used to avert constipation. Cannabis indica is recommended by many authors, but opium is far more trustworthy, and, if kept under the control of the physician, may be used for some days, or even two or three weeks, without ill consequences. Cocaine is not more useful, and has disadvantages of its own. Menthol is said to be very efficacious in relief of stomach or vomiting, even if due to organic disease; one or two grains are given in cachet with sugar of milk. As the digestive act itself is, by the nature of the case, supposed to be fairly normal, no acids, alkalis, bitters, and the like are requisite. Nor in my experience is rectal feeding, useful in many stomach diseases, required in these; although a clyster of water as hot as it can be borne may relieve the pain. For the same reason I do not trouble myself much with pepsin, pancreatin, or predigested foods. Although neurasthenics have slow digestion, and tired functions on all sides, the stomach can deal with tender and bland articles of diet well cooked and divided, and given in small quantities; and they are more acceptable to the patient and

enticing to his gastric functions than peptonised foods. Pawlow's experiments prove that a pedantic rule of diet in these cases is to be deprecated; even whims must be regarded, possibly welcomed. Of other remedies, warmth and even mild counter-irritation to the epigastrium are profitable. After a few days silver, either as the nitrate or the oxide in pills, will be of service in combination with the opium; or, if this drug be no longer necessary, alone. As the gastralgia subsides the patient must be re-edified in the usual way. Arsenic must not be forgotten as one of the best remedies for chronic gastralgia; its value in asthma, in eczema, and even in angina pectoris, probably depends on the same virtue, whatever it may be; still during the acuter phases opium is often indispensable. If there be such a thing as malarial gastralgia, of which I think I have seen one definite case, quinine must be the means of cure. I have not found strychnine of much service in any form of the malady. That the epigastric pains of gout are gastralgic, in the sense in which we are now using the name, is improbable; but if gout or hysteria be concerned in the matter, the treatment appropriate to these diseases must be applied. In the article on "Dilatation of the Stomach" (p. 543) I refer to pulmonary gymnastics as very important in certain cases in which irritable stomach or bowel is associated with defective expansion of the chest. With the means of restoring the general health we cannot occupy ourselves here.

For anorexia nervosa there is but one cure, namely, bed, isolation, and feeding by a judicious nurse not of the family. Were it not that I have succeeded more than once in compassing it, I should say that the cure of such a case ought not to be attempted at home, and that the patient should in all circumstances be removed to the care of strangers. In any case a trained nurse is indispensable, and it is also indispensable that she have her own way undisturbed by the interferences and opinions of the family and friends of the patient. If these conditions cannot be secured the physician will not desert his post, but, while doing his best, he will frankly disclaim all responsibility for the failure which will probably be his portion, and that of his patient.

Of all these cases the most difficult are those of neurotic vomiting. These patients, too, are better removed from home, but removal is by no means the almost certain cure that it is in nervous anorexia. Were it easy to prescribe the means of cure there would be less of the difficulty which I have indicated; food, however judiciously administered, too often returns, whether it be given in the smallest doses or given, on Watson's plan, in larger quantity once in twenty-four hours. The irritability of the stomach prevents the very means we desire to use. Massage without generous diet does but exhaust the patient; lavage, which I have carefully tried, is of little use; drugs are rejected. On the whole, the best means are rectal feeding, with the use of sedatives, such as menthol, or heroin hydrochloride subcutaneously, to calm the stomach and to establish tolerance of food. Here again the hypodermic use of morphine, as we know, is to be avoided if possible; moreover, in many of these cases, as

its first effects pass off, it sets up some nausea of its own. I have found some advantage from the bromides with hydrocyanic acid or, if this fail, with chloralamide, a combination which, if administered in small and repeated quantities, often soothes the irritable coats so that food is retained. Chlorobrom may be effectual. Of oxalate of cerium I have no great experience; but if it is to be of any value, it must be used in much larger doses than those usually ordered, namely, in 5 to 10-grain doses. Bismuth, in small doses of 10 to 15 grains, is valueless in these cases, or usually so; it has been used recently in doses of $\frac{1}{4}$ to $\frac{1}{2}$ an ounce mixed with a large quantity of water as an irrigation, but I have no experience of the plan, nor does it seem promising. Of arsenic I can say more; with half-drop doses in half a teaspoonful of water I have seen the stomach quieted in not a few severe cases of neurotic vomiting. It is best that these patients, if not kept altogether to bed, should lie down after food, with perhaps a hot bottle to the epigastrium; and, as nausea or disposition to vomit comes on, the patient, in spite of efforts to the contrary, must be dissuaded from raising the head. After trial of all such means without success the patient may take a quick turn and recover spontaneously; perhaps, indeed, this is the issue of most cases of neurotic vomiting.

The only remark I need make about diet is that alcohol is a dangerous remedy to recommend to neurotic persons; fortunately they do not find much benefit from it. In moderate quantities, however, with meals only, as for instance a little champagne and mineral water, it may be valuable during the worst phases of ill-health, and on complete convalescence may be pretermitted. The effects of tea, coffee, and tobacco on these patients should be carefully watched, as they, or one or other of them, may be injurious; and the odd reactions of some persons to certain foods, such as eggs or shellfish, must not be forgotten. The recreations of amusements, change of scene, and the like will not be forgotten in convalescence. Spa treatment is of little or no service in these gastric disorders, but change of air and scene may have their benefits.

Other Abdominal Neuralgias.—The difficulty in dealing with some other abdominal neuralgias, which undoubtedly exist, lies in the difficulties of their diagnosis. Although, on the one hand, the subsequent history of a patient in whom such pains have been set down as neuralgic may establish the correctness of the opinion, yet certainty in many of them cannot be predicated until after a certain lapse of time. For in some the existence of a band of adhesion, of a latent calculus, of some disordered and acrimonious secretion, of an ulcer, of some unsuspected baneful article of diet, of latent cancer, or indeed of a benign growth or aneurysm causing pressure on the branch of a nerve, may originate pain which it would be an abuse of terms to call neuralgic, at any rate in the clinical sense. Again, we may have to deal with that source of wearisome pain which is attributed by Glénard to a fall of the viscera (*vide* art. "Visceroptosis," p. 860). There is little doubt that persistent uterine

pain is often set up by dragging of this kind in neuralgic subjects. Probably visceroptosis in minor degrees does not cause pain in persons whose nervous system is stable; yet as, even in the cases of nervous instability, the distress is dependent on mechanical causes, and consequently its treatment on mechanical means, they are not conveniently discussed in this place. However, the effects of a well-fitting abdominal belt, adjusted while recumbent, will help in the diagnosis. As I have said in respect of gastralgia, so of other neuralgias of abdominal viscera, it may be that adhesion of an organ such as the gall-bladder to the parietes may occasion considerable pain of a paroxysmal or periodic character.

Still, all deductions made, it seems probable that there are cases in which visceral pain is purely neuralgic in nature; it is either independent of local disease, or, if called up by some transient catarrh or other tide of local disorder, this bears so slight a proportion to the pain as to be almost negligible in diagnosis, though not in treatment; I say "almost," because the thesis might be sustained that all neuralgias depend upon local determinants, however fugitive. That a diagnosis of abdominal neuralgia may, however, be correct sometimes was perhaps sufficiently proved by the case of a lady who called upon me in 1897. She reminded me that she had consulted me twenty years before for attacks of violent pain in the region of the liver; that in consultation with her own medical man we had decided that her pain was not dependent on gall-stone or local troubles, but was purely neuralgic (of the grounds of our decision I have no notes). She added that there was no doubt of the truth of the opinion, as the attacks had gradually diminished in frequency as her general health became stronger, but that she still had one or two attacks a year, usually after some chill, fatigue, or other depressing cause.

Hepatalgia.—In my book on Visceral Neuroses I expressed my opinion that "hepatalgia," whether the pain be actually in the liver itself or in its appendages, may be a true neuralgia, and may exist apart from such local causes as we are accustomed to enumerate; and in support of this opinion I am able to quote Pariser, who, in commenting upon 7 cases of "nervous hepatic colic," says: "This malady is usually mistaken for gall-stone colic, and in one case an operation was performed under this erroneous impression. Fürbringer thinks that in hepatic neuralgia the most intense pain is localised in the liver, and is not radiating, and although this is true in many cases, exceptions do occur." The attacks, Pariser says, last from a few minutes to a few hours, and, as in biliary colic, may end in vomiting. He defines the disease as visceral neurosis with a neurasthenic or hysterical basis, and treats it accordingly. To distinguish mere hepatalgia from gall-stone must often be impossible, even in patients of neurotic history; for the neurotic habit gives no immunity from gall-stone. In hepatalgia the liver is not enlarged, but it may be tender, and presumably the seat of the pain is in the capsule.

Enteralgia is not an infrequent disease, and is a very terrible one;

it is perhaps the most terrible of all the neuralgias. Whether the pain lie always in the bowel itself, as in the colalgia of which I have spoken already, or may arise elsewhere in the abdomen, is for the present an insoluble question. In many cases no spasm of the colon is palpable. It is usually a piercing, agonising and prostrating pain leading soon to symptoms of the incipient collapse which is at hand in all abdominal neuralgias, presumably because they tend to promote dilatation of the abdominal veins. It is perhaps commoner in men than in women, or at least as common. In my little book I recorded several cases in men which I need not repeat here. Like gastralgia it is often associated with spinal neuralgia, so that the patient is racked by pain along the course of the associated spinal nerves as well as within the abdomen itself; therefore knee-jerks, the vertebral column, the aorta, etc., must be examined even more suspiciously than in gastralgia. One of my cases, a lady for some years subject to gastralgia, lost it only to fall a victim to the worse evil of enteralgia.

In not a few instances in women there may be a difficulty in discriminating between enteralgia and pelvic neuralgia; indeed, the confusion may at times be real, for neuralgia of the pelvic parts in women may precede or accompany enteralgia. A lady of some thirty years of age, whom I saw with Mr. Day of Baldock, was by such a pain in the right hypogastrium gradually debarred from the habits of a very healthy active outdoor life. I could scarcely admit that she was a "neurotic," not even a "quiet neurotic," and the conditions of her life were happy and wholesome; yet the most searching and intimate scrutiny (with and without anaesthetics) failed to reveal any local cause. After two or three years of this she had a sudden attack of articular gout, on which the pelvic pain promptly and completely vanished. It is pretty certain that enteralgia is associated more definitely with gout than are the other abdominal pains, though no doubt there are many cases of enteralgia which own no such cause. For instance, a gentleman I saw some years ago with Mr. Holmes of Leeds presented the following series of nervous miseries: first, he had suffered from cervico-brachial neuralgia with hyperaesthesia of the scalp and Valleix' points; at a later date he suffered from insomnia; later, again, from a strange sensitiveness of the skin to cold—the slightest draught, such even as the wafting of the leaves of his ledger, being painful to him; then periodic coryzas, with extreme defluxions and asthmatic dyspnoea, fell upon him; and, finally, with no less vehemence, he took to enteralgia. He was much benefited by a long rest with the systematic use of sea-water baths. I have never found electricity of much service in any of these cases; it gives no immediate relief, and perhaps we are not faithful and patient enough to persist in its use.

In the diagnosis of enteralgia we are less embarrassed by the alternative of gall-stone, the resemblance to this colic is not great; enteralgia usually begins near the navel, is more stabbing than gall-stone, and takes larger excursions not only about the belly, but, as I have said, in the

courses of the neighbouring spinal nerves. The belly may be blown out, even with open bowels; or again, and more frequently, it is retracted and tense. Spasm of the colon I have anticipated already (p. 399). The behaviour of the bowels is often irregular, irritable or obstinate; membranelike shreds, if present, must not be overlooked (*vide* Membranous Colitis, p. 816). The modern methods of analysis of the faeces will probably be of great assistance in the discrimination and cure of maladies of this class. One malady there is which must not be confounded with enteralgia proper; namely, the pain in the region of the hepatic flexure of the colon which haunts the victims of melancholia or hypochondriasis. This pain is never acute, it is rather of a wearing and depressing character; moreover, it needs quite different treatment. The blue pill and black draught which relieve the hypochondriacal pain would aggravate that of enteralgia. Arsenic is not the sure friend in the other visceral neuralgias that it is in gastralgia, but it is not without value. Belladonna, or rather atropine by the skin, is of considerable service in the spasms of the colon, but is only moderately helpful otherwise. I ought perhaps to emphasise again the warning against the risk of permitting any of these abdominal neuralgiacs themselves to get hold of opium in any of its forms; the relief is as sure as the pain they suffer is intense, but the almost certain subsequent abandonment to the drug means physical and moral deterioration. (*Vide* art. "Morphinism," Vol. II. Part I. p. 946.)

If, then, we cannot deny them the gift of opium from time to time, we shall counsel these sufferers to submit patiently, persistently, and hopefully to a methodical course of diet, hygiene, and such medicines as, by removing the causes that in each case may appear to be operative, and by raising the state of the nervous system and of the blood, and the nutrition of the tissues to the highest point, will place them above the reach of these perturbations. I should remind the reader that evidence of lead must be carefully sought for in all cases of enteralgia; and that in all abdominal pains scrupulous search must be made for the minutest umbilical hernia.

Nephralgia is said to be the commonest of the abdominal neuroses, and the one of which we should know most; it is true that in quest of stone not a few neuralgic kidneys have been opened in vain. That one kidney may be the seat of a grievous pain of a "functional" kind supports the hypothesis of the possibility of pure neuralgia in other abdominal viscera, such as the liver and stomach; though it cannot be definitely asserted that the source of the pain actually lies in the kidney itself, its apparent origin. To distinguish the renal colic of gravel, oxalates, and the like from mere nephralgia may be practicable if sufficient attention be given to the urinary deposits; but to distinguish the pain due to a fixed stone is too often impossible. Nausea or vomiting may coincide with either affection, and even the appearance of blood in the urine is no crucial fact, strange as this may seem; on the indication of haematuria many an aching kidney has been opened and found

empty of stone. Some authors appear to find an explanation in calling such haemorrhage "angioneurotic"; and the pain may be due to tension of the capsule in fits of vascular distension, which sometimes issue in haemorrhage. Mr. Reginald Harrison has thrown some light on this dark subject by his observations on high renal tension. Dr. Goodhart reports a remarkable case in which a necropsy was made with negative results; in another important case, also with haematuria, in a lady, a medical colleague joined me in opposing the surgeon's interference, who, however, triumphed, not in the discovery of stone or other local disease, but in the cure which followed his apparently abortive incision; another in which operation was repeatedly performed is reported by Prof. Howard Marsh. Yet, as I have already said, a neurotic habit in the patient herself or in her family does not prevent lithiasis; indeed, some observers suggest that such persons are rather liable to this perversion. And the operation of nephrotomy, if unsuccessful as regards its primary purpose, is not infrequently the means in some obscure way of relieving the pain, at any rate for a time. So grievous is nephralgia, so futile, as a rule, are the means in the hands of the physician, and so safe are operations nowadays, that an exploratory operation may be justifiable in doubtful cases, even if there be no definite evidence of stone or gravel, and no haematuria, as indirectly relief may accrue from the incision. Apart from the operation there is nothing to be done, in many cases, but to treat the general condition of the patient by improved nutrition, massage and the like, in order to enable the re-created system to throw off the burden: I say in many cases—for in some an aberrant function may be detected, and perhaps corrected with relief. There may be a high acidity of the urine; or indeed a deposition of crystals, however fine in grain or minute in quantity. Oxalates, again, may be the source of the irritation; and persons of a neurotic bent are victims to recurrent renal pains from causes such as these which, in ordinary persons, would escape notice or set up but a trifling discomfort. In *Visceral Neuroses* I report a case in which a fine crystalline deposit (of uric acid, if I remember right) was repeatedly found in the urine of a lady who had long been subject to a distressing nephritic colic. It may be, indeed, that some aberrancy, of so trivial a degree as to escape recognition, lies at the bottom of all renal colic, and an appropriate course of mineral waters may dispel the pain; on the other hand, the ordinary means of combating lithiasis and of rectifying the functions of the kidney too often fail to relieve the sufferers.

In concluding this section, I cannot altogether overlook the extraordinary statements of certain French physicians concerning the effects of hypnosis in cases of gastric and other neuralgias. Sollier, for example, declares that by inducing periods of hypnosis he can cure and recall anorexia nervosa, and other such functional disorders, at will—the patients being conscious of favourable revolutions in their viscera. According to these accounts, maladies, like kings, can be made and unmade at pleasure; and by these proceedings the author declares

himself able to carry out many experiments on nervous dyspepsia and other stomach disorders. It is my duty to allude to these claims, and the efficacy of hypnosis in nervous maladies is supported by something like scientific proof; but this field of experimental therapeutics is too large a one to enter upon here.

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GASTRITIS

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Acute Gastritis.—Syn.: *Acute Gastric Catarrh.*—Inflammation of the stomach is divided, according to its course, into acute and chronic; according to its pathology, into mechanical, catarrhal, toxic, symptomatic, diphtheritic, and phlegmonous.

Etiology.—Inflammation of the stomach, like inflammation of any other organ, may be due to mechanical injury, thermal or chemical irritants, or invasion by living organisms either animal or vegetable. Amongst the mechanical causes is to be reckoned, in the first place, excess in food, as when people eat too heartily after a long fast, or are tempted by palatable food and especially by unwonted delicacies; a second cause is an injurious quality of food, which may be either too rough, hard and indigestible, or may be undergoing such changes of fermentation or decomposition as lead to the formation of powerful

irritants. Next come articles of drink, and more especially alcoholic liquors. These may irritate the stomach either (i.) by the amount of alcohol they contain, or (ii.) by the acid which is either originally present in some of them, as for example in certain wines, or is formed from the alcohol in the stomach by acetic fermentation. In many cases the effect of excessive food and excessive alcoholic indulgence are combined to produce acute gastric catarrh.

Poisons, such as acids, alkalis, or salts of the heavy metals, all cause acute inflammation of the stomach; and amongst the mineral poisons one of the most common and powerful is arsenic. Certain organic poisons act as powerful irritants to the stomach, and amongst these may be reckoned some of the products of albuminous decomposition. Some of them may act, either (i.) by direct introduction into the stomach, or (ii.) by excretion into the stomach by its mucous membrane after their formation in the intestine, and elsewhere, and subsequent absorption into the circulation. Antimony when injected into a vein causes vomiting, in part at any rate by its direct action on the stomach; and not by affecting the central nervous system as formerly supposed. It is carried by the blood to the stomach, there excreted, and subsequently exerts a local action on the stomach itself. Many organic substances are excreted in a similar way. Cobra venom was found by the late Sir J. Fayrer and myself to be a powerful local irritant when applied to the stomach of a frog. It seemed to me probable that it would also be excreted by the gastric mucosa, and this was found by Alt to be really the case. Some toxalbumins are excreted by the stomach in cases of cholera also, and have been found by Alt in the vomited matter. The vomiting which indicates irritation of the stomach at the beginning of many acute diseases is not improbably due to the excretion of toxalbumins or albumoses; and the same is probably the case in the vomiting of patients suffering from renal disease. It is probable, therefore, though not yet certain, that the vomiting symptomatic of gastric irritation in many infective diseases is really of toxic origin.

All the symptoms of gastric catarrh frequently occur after great emotion, and especially after distressing emotions, such as anxiety, sorrow, fright, or anger. It is impossible to say how far these symptoms are due to the direct action of the nervous system on the stomach, and how far to its indirect action in altering the processes of digestion and giving rise to the formation of more or less poisonous products which subsequently act as gastric irritants. The same may be said of external cold as a cause of acute gastric catarrh. In many persons, general exposure to cold, or sitting with wet feet, will bring on gastric catarrh, just as in others it would cause catarrhal inflammation of the trachea and bronchi. Catarrh of the stomach not infrequently follows catarrh of the respiratory passages. In some people it occurs as the respiratory catarrh is passing off, and in popular language the cold in the chest is said to have been carried off by the stomach and bowels. In some cases the swallowing of mucus from the respiratory passages, and

especially from the naso-pharynx, gives rise to gastric catarrh; and it is not infrequent in phthisical patients who swallow the sputum instead of expectorating it. Amongst the organisms found to have been a cause of acute gastric catarrh are the yeast fungus, anthrax bacilli, the favus fungus (*Achorion schoenleinii*), and the *Oidium albicans*.

Inflammation of the stomach may occur from the presence of animal parasites, such as the weevils of cheese and the larvae of insects.

Gastric catarrh occurs also in association with organic disease of the stomach, such as stenosis of the pylorus, whether simple or malignant, and malignant disease of other parts of the stomach.

Pathology.—Our knowledge of the slighter stages of acute gastric catarrh is derived almost entirely from the observations of Beaumont on the stomach of a young Canadian voyageur, Alexis St. Martin. In consequence of a severe wound in his side from a charge of duck-shot, his stomach became adherent to the abdominal wall, and an opening existed in it, usually covered by a fold of mucous membrane, which, when pushed back, revealed the interior of the stomach. Beaumont describes the inner coating of the stomach, in its natural and healthy state, as being of a light or pale pink colour, varying in its hues according to its full or empty state. "It is of a soft or velvet-like appearance, and is constantly covered with a very thin, transparent, viscid mucus lining the whole interior of the organ. Immediately beneath the mucous coat, and apparently incorporated with the villous membrane, appear small spherical or oval-shaped granular bodies, from which the mucous fluid appears to be secreted." With a thin whitish fur on the tongue and a rather craving appetite, "several red spots and patches, which were tender and irritable, appeared over the inner surface of the mucous membrane." When the countenance was sallow, the tongue covered with a thin white coat and the appetite failing, the stomach presented several deep red patches on its inner coat. When sick-headache was present, with pain and uneasiness of the stomach, general debility, lassitude, a depressed pulse, dry skin, coated tongue, and costiveness, the stomach presented numerous white spots, or pustules resembling coagulated lymph spread over its inner surface. After indulgence in spirits—with a thin, yellowish-brown fur on the tongue and uneasy sensation and tenderness at the pit of the stomach, vertigo, dimness and yellowishness of vision on stooping down and rising again, and a sallow countenance, but no general discomfort nor failure of appetite—the mucous membrane of the stomach was found to present an erythematous appearance and livid spots, from the surface of which exuded small drops of grumous blood, numerous patches of aphthae, and a thick coating of mucus; the gastric juice was also mixed with thick, ropy mucus or muco-purulent matter, slightly tinged with blood, resembling the discharge from the bowels in dysentery. The abundant and tenacious secretion from a stomach in such a condition is found by microscopical examination to consist of mucus with leucocytes and epithelial cells undergoing mucoid degeneration. The cylindrical epi-

thelium of the tubules undergoes mucoid degeneration and desquamation; and in such cases microscopical examination of the mucous membrane itself shews the peptic cells to be loosened and granular, and the epithelial cells at the mouth of the tubules to be soft, swollen, filled with mucus, and desquamating. The blood-vessels are distended, the tissues are filled with leucocytes, the submucous tissue is somewhat oedematous, and there may be slight haemorrhage from spots on the mucous membrane such as those seen by Beaumont.

Symptoms.—In acute gastritis, when caused by powerful poisons, there is generally severe pain in the epigastrium with great tenderness and violent vomiting, the vomited matters consisting first of the contents of the stomach, afterwards of mucus, frequently of bile, and finally of mucus tinged with blood, or even of blood more or less pure. As the poison may be completely ejected from the stomach without passing into the intestines, gastric inflammation may occur without inflammation of the intestines; but if this be not the case, and the intestines also become implicated, pain and tenderness extend over the whole abdomen, and diarrhoea more or less violent usually occurs. Even when the inflammation is limited to the stomach the circulation is usually much affected. The face becomes exceedingly pale and pinched, the surface is cold, there is abundant cold perspiration, and frequently also a profuse flow of saliva. The pulse may be quick or slow, but is generally feeble. In the case of a powerful poison, these symptoms are apt to remain more or less persistently for several days. When the irritation is less powerful, as is the case when the stomach has been simply overloaded with food or drink, or when the food or drink has been of an unsuitable nature, the mere evacuation of the stomach by vomiting gives much relief, and the patient may feel almost well; although a certain amount of weakness and lassitude usually remains behind. If the vomiting is at all violent the tenderness in the stomach may continue for some days; and sometimes a soreness, which is probably due to straining of the oesophagus, is felt all down the chest behind the sternum for a day or two. This rapid disappearance of symptoms usually occurs, however, when the irritant has been applied for a short time only, and when the mucous membrane of the stomach has not been much inflamed. When the mucous membrane itself is inflamed certain symptoms remain after the evacuation of the strong irritant to which the inflammation is due.

These symptoms may also appear more gradually in consequence of the continuous action of a slighter irritant; in which case, instead of beginning with violent vomiting and pain, which subside to a certain extent after the evacuation of the stomach, the symptoms go on gradually increasing for days or weeks, until they may culminate in great pain and vomiting such as has just been described. These symptoms usually are a thickly furred tongue and loss of appetite, frequently accompanied by a good deal of thirst. Not only is there no desire for food, but eating may bring on a feeling of nausea, and nausea may

arise at the mere thought of food. Frequently, however, although ordinary food is distasteful, there is a craving for strong-tasting and savoury things, such as salt fish, pickles, curry, Worcestershire sauce; and occasionally the appetite may be somewhat craving rather than deficient; but this craving appetite is usually soon satisfied by even a very little food, and if more food be taken it turns to loathing.

The stomach is tender on pressure, and, being distended with gas, is often prominent in the epigastrium. Gaseous eructations are frequent, and may be accompanied by sour fluid so acrid as to burn the throat, to set the teeth on edge, and to give rise to much discomfort at the lower end of the ensiform cartilage—at the point corresponding to the junction of the oesophagus with the stomach. In some cases hiccup is a persistent trouble, and there is a feeling of weight and pain in the epigastrium which seems to pass through between the shoulder-blades. The bowels are costive unless the catarrhal condition extends to the intestines as well. This is to be anticipated as very little food is taken or absorbed. The urine is generally scanty, high-coloured, and deposits a brick-dust sediment. Generally also there is a feeling of weight in the head, and headache, frontal, temporal or occipital, is frequent; sometimes patients complain that the pain feels like a ball inside their head. There is difficulty in concentrating the thoughts; all exertion, mental or bodily, is distasteful, and the patient is very low-spirited and not infrequently irritable. The temperature is often normal, but in some cases it rises as high as 102° or 103° F.; this rise is usually preceded by a rigor.

Diagnosis.—The symptoms of acute gastric catarrh are so distinctive that usually little doubt can exist regarding the nature of the attack; but it is not always easy to say whether it may not be due to the presence of toxins or to the invasion of pathogenetic organisms, such as typhoid bacilli, which may affect other organs as well.

If the attack be not accompanied by a febrile temperature, and there be a clear history of some dietetic error or exposure to a chill, the probability is that the symptoms are due to acute catarrh only; but if the temperature be high the attack may denote the onset of some infective disease, and a diagnosis can only be arrived at by watching the case for some days. Gastric catarrh is most likely to be confounded with commencing enteric fever, but in the latter disease the rise of temperature is less abrupt, the remissions are more marked, and the duration of the fever is more prolonged. Cases of acute gastric catarrh usually end in recovery in one or two days, although they may last for as long as a week or perhaps even for a fortnight. Such prolonged cases may well be mistaken for enteric fever; but in addition to the diagnostic points already mentioned, the spots are absent and the spleen is not enlarged.

When the attack sets in with severe headache and delirium, it may be mistaken, especially in children, for meningitis, or for the beginning of measles, or of scarlet fever; but the course of the disease and the absence of the rash after its proper time enable a diagnosis to be made.

The treatment of acute gastric catarrh consists first in removing all

irritant substances from the stomach; and, secondly, in soothing the inflamed organ. The stomach naturally tends to eject any irritating substance present in its cavity; but, after the main portion of its contents has been ejected by vomiting, small quantities of exceedingly bitter bile mixed with mucus may remain and give rise to constant nausea and retching. In order to remove this it is advisable to administer several tumblerfuls of lukewarm or of warm water, and, after this has been vomited, to repeat the process several times; thus all irritant substances are washed out of the stomach, and after this has been done two or three times the vomiting will often cease. Occasionally the first draught of two or three tumblers of water will so dilute the contents of the stomach that the irritation is no longer strong enough to produce vomiting. In such a case it is better to tickle the fauces with a feather, so that the water may be vomited and the stomach cleansed; or the gastric siphon may be used: but unless the patient is accustomed to the use of this instrument the plan of swallowing hot water and tickling the fauces is generally preferred. In mild cases this treatment is all that is required, and after a few hours' rest the stomach is all right again; but for at least a day afterwards the diet should be very light, consisting chiefly of milk and farinaceous food. When the attack is more severe and the catarrh has affected the bowels also, as shewn by a tendency to diarrhoea and pain not confined to the gastric region but extending over the abdomen, half an ounce of castor oil with 8 or 10 minims of laudanum should be given; so as to clear out any irritant from the intestine. If, after the oil has acted, the pain or nausea or vomiting still persist, or if there be any yellowness of the conjunctivae, half a grain of calomel may be given every half hour till 5 grains have been administered; or 5 to 10 grains may be given at night, and next morning a saline purgative, such as a glass of aperient mineral water or some effervescent sulphate of magnesia, sulphate of soda, or phosphate of soda.

The best food is simply milk diluted with one-fourth to one-half its volume of soda-water; unless the bowels be loose, when lime-water should be substituted. This dilute nourishment may be given in quantities of 5 or 6 ounces every two hours; but should it be rejected by the stomach, it is well to let the patient take nothing but a little iced water and swallow small pieces of ice until the acute irritation has subsided. If there be any fear of failure of strength, the nutrition may be maintained by nutritive suppositories or nutritive enemas. After the irritant matters have been evacuated from the stomach, one of the best sedatives is bismuth, which may be advantageously given according to the following formulas:—℞ Bismuth. carb., sodii bicarb. āā gr. x., spt. chloroformi ℥x., aquae menthae pip., vel aq. cinnamomi, vel aq. flor. aurant. ℥j. M. Ft. hst. ante cibos sumend. If there be much pain, 5 minims of tincture of opium or liquid extract of opium may be added to each dose; and if the vomiting be severe 5 minims of dilute hydrocyanic acid may be added, either with or without opium, as the case

seems to demand. Should the bowels be loose some chalk may be added to each dose, or two drachms to an ounce of chalk mixture may be given either with the bismuth mixture or after each loose motion. If the bismuth tend to constipate the bowels two fluid drachms or more of liquor magnesii carbonatis may be added to each dose. Sometimes dilute hydrocyanic acid in an effervescing form seems to be best, and it may either be prescribed in doses of 5 to 8 minims in one or more fluid drachms of water to be added to half a wine-glassful or more of any effervescing water, or it may be given with 20 grains of sodium bicarbonate in an ounce of water to be mixed at the time of administration with 17 grains of citric acid. Counter-irritation to the epigastrium by a mustard poultice or mustard leaf tends to relieve both pain and vomiting; and a full warm bath is often very soothing and useful, both in the case of children and of adults.

Hiccup is sometimes a most troublesome complication. Many remedies have been advised for it. Evacuation of the stomach, either by large draughts of hot water and subsequent vomiting, or by the gastric siphon, may be useful, and remedies subsequently applied have a better chance of acting. Sedatives of various kinds may be used, dilute hydrocyanic acid, morphine, chloral, bromide of potassium, or a combination of these. I have also found phenalgin useful in $7\frac{1}{2}$-grain doses every three hours.

Chronic Gastritis.—Syn.: *Chronic gastric catarrh, Chronic dyspepsia.* — **Etiology.** — The causes of chronic catarrh are the same as those of acute catarrh; and attacks of acute catarrh, especially if frequently repeated, pass into a chronic condition.

The irritation of the stomach which produces chronic rather than acute catarrh is usually less intense in degree, and frequently repeated. In acute catarrh the very violence of the irritation causes ejection of the irritant, while in chronic catarrh, the irritation being less severe, the irritant remains in the stomach, producing perhaps no more than discomfort at the time, but gradually altering the mucous membrane and even the muscular coats. One of the most common causes of common gastric catarrh is free indulgence either in acids or substances which yield acids in the stomach. Thus, among the peasantry of Southern Europe sour wine is a frequent cause of gastric catarrh; and all wines, even those which taste sweet, are strongly acid to litmus-paper. Spirit, when taken in a concentrated form, will act as a powerful irritant; when more dilute, it frequently undergoes acetic fermentation, and thus acts as an acid. Sugar, especially when taken in a concentrated and soluble form, is apt to give rise to acidity; while starches, being less soluble, are generally passed through the stomach without undergoing such a change, although if the stomach be dilated they also may undergo fermentation. Fats are apt to undergo change also and to yield butyric acid, which is one of the most irritating of all acids to the stomach. But people frequently forget that a rancid fat, which would make them sick if taken by

itself, does not lose its irritating properties when it is mixed up with flour and made into pastry (*vide* p. 364). Another cause is eating too quickly; another is drinking much liquid with meals. Both of these practices tend to interfere with the rapid digestion of food, and thus to allow time for fermentative processes to go on. Retention of food in the stomach with consequent fermentation and chronic gastritis occurs when the stomach is dilated, in consequence either of atony or of pyloric obstruction due to cicatricial contraction or to new growths. Malignant disease in other parts of the stomach may directly give rise to catarrh. Obstructed circulation through the stomach tends to induce chronic catarrh; thus, it is frequently found associated with conditions which obstruct the gastric circulation; such as cirrhosis of the liver, phthisis, and mitral disease, obstructive or regurgitant.

An altered condition of the blood, such as anaemia, chlorosis, renal disease and gout, may lead to chronic catarrh.

Pathology.—It has already been mentioned that in acute inflammation mucoid degeneration of the cells in the gastric glands and mucoid degeneration and desquamation of the epithelium occur. In chronic catarrh the same processes take place, their long continuance leading to wasting of the mucous membrane with increased formation of connective tissue and a tendency to the formation of cysts in the tubules. Sometimes the atrophy occurs fairly evenly over the whole surface of the stomach; but at other times cystic degeneration occurs to a large extent in certain parts, so that the mucous membrane, instead of being smooth and thin, rises in folds and knobs, or polypi. For this reason it has been described as "gastritis polyposa." In the cases just mentioned the change chiefly affects the mucous membrane, and there is no very marked increase in the connective tissue. The connective tissue in some cases of chronic disease of the stomach is very little altered; in others it undergoes great increase. The increase may take place both in the upper and lower layers of the mucous membrane, and in some cases the increase of connective tissue is so great that the walls of the stomach are greatly thickened and the stomach itself diminished in size. To this condition the name "cirrhosis" has been given (*vide* p. 437).

Symptoms.—As in acute catarrh, chronic irritative conditions of the stomach may be accompanied by a craving appetite, or by loss of appetite. The tongue is usually coated, there is weight, distension, uneasiness, oppression or pain in the epigastrium; a good deal of flatulence; sometimes a disagreeable taste in the mouth; frequently tenderness on pressure. The pharynx is often congested; there may be a tendency to hawk and spit, and sometimes there is a tickling, irritating cough, which may come in severe paroxysms. Eructation of sour fluid and pain at the pit of the stomach are also present, such as have been described already under dyspepsia. Headache, generally frontal, is often complained of, and there is indisposition for any exertion mental or bodily, depression of spirits and often irritability of temper. In some cases there is distinct giddiness or palpitation.

Diagnosis.—Three forms of chronic gastric catarrh have been described by Ewald, namely, simple, mucous, and atrophic. In the first two hydrochloric acid is diminished, but the normal ferments are present, and the secretion of mucus is scanty; in the second form the mucus is abundant. In the third form both hydrochloric acid and the characteristic ferments of the stomach are absent. The methods of ascertaining these conditions have already been given (*vide* p. 279).

Primary atrophy of the stomach has been described by Fenwick. Its symptoms are progressive weakness and anaemia, with a sallow colour almost exactly like that of pernicious anaemia. This condition comes on very gradually without any apparent cause, generally attacks persons past middle life, and on post-mortem examination the gastric mucous membrane has been found pale, generally thin, and adherent to the submucous tissue. The gastric glands are firmly united and distended with cells and molecular matter. At a latter stage the tubules disappear completely, or sometimes a number of flask-shaped bodies loaded with cells remain at the bases of the tubules, while in the rest of the mucous membrane no glandular structure can be recognised.

The diagnosis between chronic gastric catarrh and malignant disease is often quite impossible. If the symptoms persist in spite of treatment, but the patient is under forty, the most probable diagnosis is chronic gastric catarrh; if the symptoms persist in spite of treatment in a patient over the age of forty the presence of malignant disease is exceedingly probable. In a case of long-standing chronic catarrh, it is not until the presence of a tumour becomes evident that the diagnosis of malignant disease can be made with certainty. Malignant disease indeed may come on in persons who have long suffered from the symptoms of chronic gastric catarrh. One of my patients had been accustomed to wash out his stomach for three years, and not until after the lapse of this time did it become evident that he was suffering from malignant disease. In another patient, who suffered for fourteen years from dyspepsia, the stomach had been dilated for at least nine years, but only five months before death did a tumour become evident.

In order to distinguish cases of chronic atrophy of the stomach from pernicious anaemia, the attention should be directed to the presence or absence of the gastric ferments and of hydrochloric acid in the stomach, to the presence or absence of megaloblasts in the blood, and of amines in the urine.

Treatment.—The first essential in the treatment of chronic gastric catarrh is to remove the sources of the irritation which brought it on. The general dietetic treatment has already been given under "Dyspepsia," and I may sum it up shortly by saying the patient must avoid alcohol, sugar, and condiments, eat fat sparingly, chew slowly and masticate thoroughly. Liquids and solids should not be taken together; and, if necessary, farinaceous and protein foods must be taken at different times. The body should be kept warm, the mind easy, and the bowels open. In cases of dilatation, great relief is afforded by washing out the

stomach. This is best done in the morning, so that the long night's rest may allow as much food as possible to be absorbed, and the waste of washing out the stomach be as small as possible. (For further information on this subject *vide* p. 550.) Water just slightly coloured with permanganate of potash may be used instead of plain water; or two to five grains of boric acid may be added to every ounce of the water.

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L. B

Phlegmonous Gastritis.—This is a severe inflammation passing on to purulent infiltration of the stomach wall. Its main incidence is on the submucous coat, though it usually affects the other coats also to some extent; thus, as well as by its severity and gravity, it is distinguished from ordinary inflammations of that organ which mainly attack the mucous coat, and are generally spoken of as catarrhs. Fortunately it is rare. The total number of cases recorded up to the present time number a little over 100; of these 15 have occurred in this country. It is met with in two well-marked anatomical forms: (A) the diffuse, and (B) the circumscribed; in the former the suppurative process is

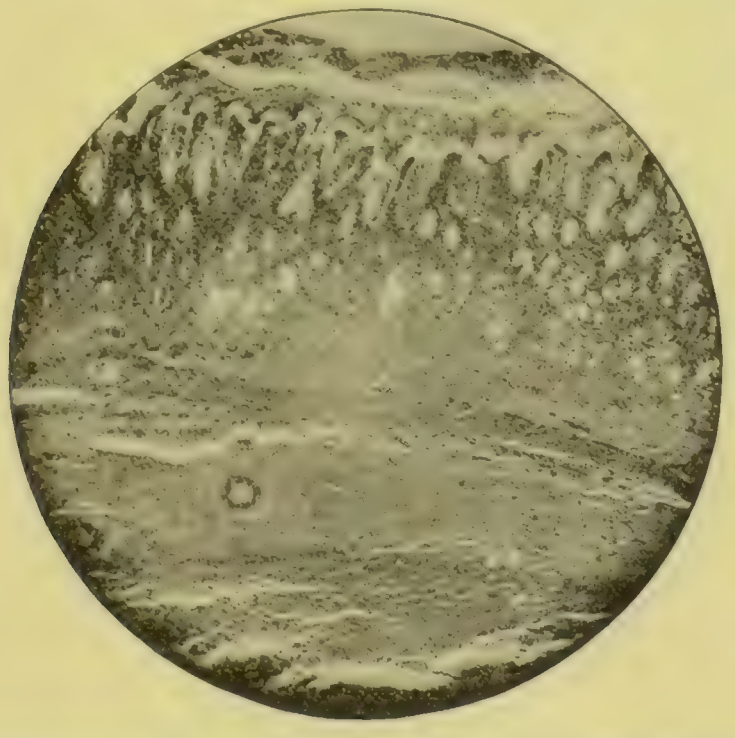


Fig. 13.—Photomicrograph of a vertical section of the mucous and part of the submucous coats of the stomach. × 35. The round-celled invasion of both coats, particularly of the submucous, is seen.

more or less widely and diffusely spread throughout the stomach wall, in the latter it forms a definite collection of pus in the gastric wall—in other words, an abscess. In several of the cases a combination of both forms was present.

A. **The diffuse form** occurs with nearly twice the frequency of the other.

Pathological Anatomy.—The stomach wall is invariably thickened, sometimes greatly so; even eight or nine times that of the normal. The thickening may be general and uniform but is more frequently local, the pyloric region being its usual seat. The capacity of the stomach, as a whole, is sometimes considerably enlarged, often unaltered,

rarely diminished. (*a*) The mucous coat is most frequently swollen and hyperaemic, sometimes it even presents haemorrhages. In a fair number of cases, though swollen, it is pale throughout; in a few it seems to be normal. The morbid appearances may be continued for a short distance into the duodenum. It is not a little remarkable that the continuity of the surface is commonly unbroken. Slight and even deep ulcerations have been met with in a few cases; but perforations, extending into the submucous coat and allowing pus to be expressed on squeezing, have been observed in five or six cases only. Microscopically, the chief change is an abundant round-celled infiltration between the gastric

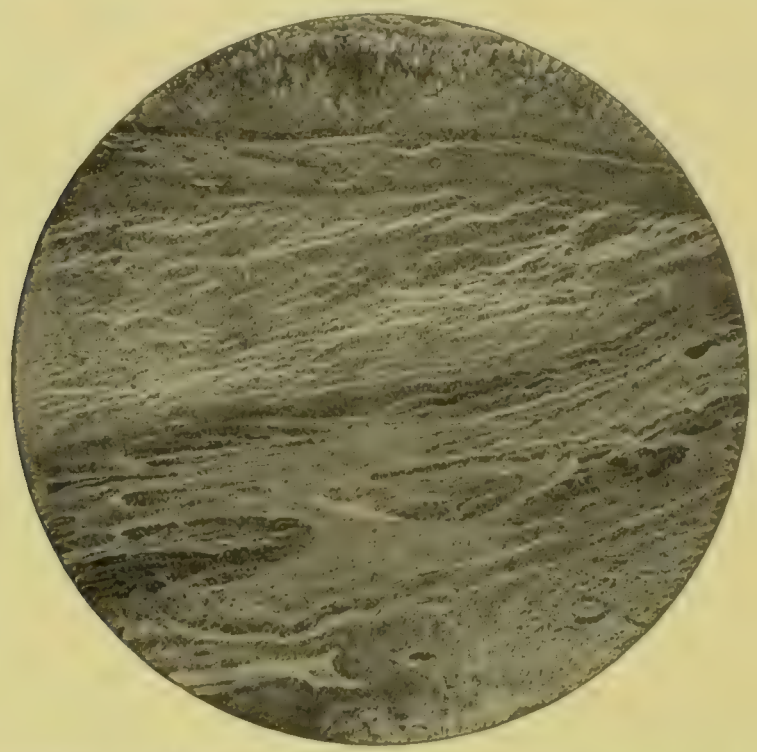


FIG. 14.—Photomicrograph of a vertical section of the stomach in the pyloric region. × 10. The submucous coat shews extensive cell-invasion (pus), particularly towards the left. The other coats are similarly though much more slightly invaded.

glands; there may be dilatation of the superficial blood-vessels also, and cloudy swelling, extensive degeneration, or even necrosis of the secreting cells. The muscularis mucosae may be normal, or irregularly swollen in places and infiltrated with round cells (Fig. 13), or more or less necrosed. The changes are indicative of an acute pathological process. (*b*) The submucous coat.—The thickening is here extreme, though its extent varies in different cases, and often in different parts of the same case. It is white or pale yellowish-white in colour, and of a soft, diffluent, or even fluid consistence; it is rarely firm and solid. It is usually continuous over the affected part, though it may be interrupted, and may shew specially swollen foci in places. It is generally sharply bounded by the pylorus; but occasionally it extends into the duodenum

for a short distance. In the same way it may infiltrate the walls of the lower end of the oesophagus. An oedematously swollen zone is usually seen surrounding the purulently infiltrated part. Microscopically, immense numbers of leucocytes are seen everywhere throughout its thickness, sometimes so numerous as completely to obscure the natural structure. Fibrin in the form of a close and abundant network is usually present, particularly at an early stage. It is prolonged around the blood-vessels both here and in the muscular coat. The blood-vessels are dilated and the tissues of their coats swollen (Figs. 13, 14). The disease appears to begin in the deeper layers of the submucosa.

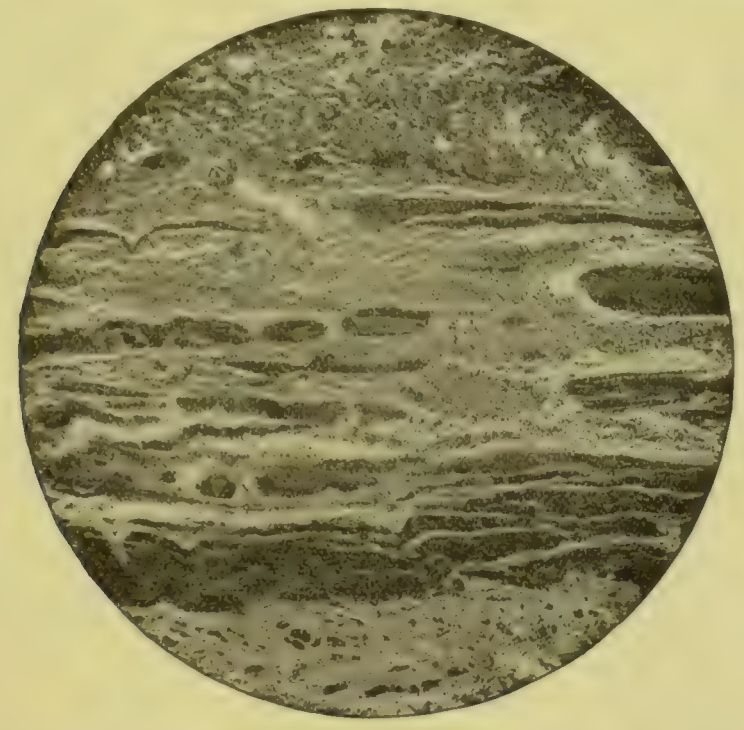


FIG. 15.—Photomicrograph of a vertical section of the muscular coat. × 35. It is invaded by cells in every direction.

Micro-organisms in great abundance, especially streptococci, have been found in about twenty of the more recently recorded cases. (*c*) The muscular coat is often little altered, sometimes swollen and oedematous, less frequently infiltrated with fibrin or sero-fibrinous pus; and in a considerable number of cases it is degenerated, necrosed, and more or less completely destroyed in whole or part, thus allowing the pus to reach the serous coat. Microscopically, a plentiful cellular invasion of the lymph-spaces and connective-tissue strands between the muscle-bundles may be seen in cases where to the naked eye the muscle appears normal; and the muscular fibres may be swollen, granular, or fatty (Figs. 14, 15). In other cases the muscular fibres are more or less completely necrosed. Micro-organisms may be seen throughout its whole thickness. (*d*) The serous and subserous coats are

unaffected in a great many cases, but an oedematous swelling or a purulent infiltration of the latter and a dimming of the gloss of the former may be seen. In some cases the inflammation of the serous coat is well marked, and exhibits the various degrees of a local peritonitis. The same micro-organisms may be found here as in the other coats. (*e*) Complications.—General peritonitis is by far the most frequent; it occurred in fully half of the cases. Among other lesions pericarditis, pleurisy, abscess of the liver, and parotitis have been observed.

Etiology.—(*a*) Sex.—The disease is much more frequent in men than in women. Oser, in a total of 27 cases, gives the proportion as 8 to 1; while in a total of 65 cases I have found it to be between 3 and 4 to 1.

(*b*) Age.—It is most frequent in early adult and middle life. I have been able to ascertain the age in 65 cases, and find 5 between 10 and 20, 18 between 20 and 30, 10 between 30 and 40, 13 between 40 and 50, 7 between 50 and 60, 7 between 60 and 70, and 4 between 70 and 80. The youngest was 10 and the oldest 85.

(*c*) Alcoholic excess has been thought to play an important part in the production of the disease, as it was present in nearly one-half of the cases collected by some observers. This proportion, however, is undoubtedly too great; one-fifth or one-sixth would be more correct: moreover, when we compare the rarity of the disease with the frequency of alcoholic excess, we cannot place much reliance upon it.

(*d*) Dietetic Errors.—In a certain proportion of cases the disease has followed immediately after a meal, so that the food has been suspected. Two, at least, of the patients habitually ate to excess after periods of fasting or spare feeding; but in none of them could a more direct connexion of this kind be traced.

(*e*) Injury.—There is a history of a previous injury to the external epigastric region in only two or three of the cases at most, and its causal influence must be regarded as of the slightest. In laparotomies for stomach troubles the disease has followed incision of the stomach wall in a few cases. The organ may be injured from the inside either by trauma or ulcer. Klieneberger blames medicinal doses of iodide of potassium as the cause in a tailor aged 67, and Mau records a case in a girl 18 years of age following upon a poisonous dose of oxalic acid. A few cases have also occurred in connexion with simple or malignant gastric ulcer, but this is very exceptional.

(*f*) Blood-Poisoning.—The great majority of cases are primary, and are thus called "idiopathic." A few are secondary to some injury or operation, puerperal fever, typhus fever, gastric cancer or ulcer, and so forth. These are called "metastatic" cases. In the idiopathic forms the infection must come from the stomach or from the blood. In the former case it must arise in (*a*) the food introduced from without; (*β*) the secretions which act upon it; (*γ*) the poisons which may be produced by the action of the normal or abnormal ferments or accidental gastric contents. Ordinary alimentary substances can hardly be thus harmful, even if we take into account individual idiosyncrasies concern-

ing shell-fish, mackerel, and the like. The ordinary gastric secretions, namely hydrochloric acid and pepsin, have been experimented with. Bouveret and others assert that the former, and Bergmann and others that the latter, is injurious when injected into rabbits, and will kill them. Trypsin may get back into the stomach; and it has been shewn by Pawlow and others to produce a local necrosis and inflammation, which may be haemorrhagic, but not suppurative. The third division includes a number of conditions as yet imperfectly known. Stricker and Kocslakoff found that pus sometimes appeared in the submucous coat of the stomach after injection of a small dose of ammonia and water; and Pilliet produced a phlegmonous gastritis by the injection of caustics (sulphuric acid and croton oil essence) which differed from that of man in being more haemorrhagic, less purulent, and free from micro-organisms. We can hardly suppose, however, that substances similar to these are formed within the stomach. On the other hand, the stomach, for a variable time after a meal, usually contains a great number of bacteria, and the entrance of one or other of these into its walls might lead to the characteristic results. The fasting healthy stomach contains no germs. The sudden onset, rapid course, and pathological appearances presented by the disease all favour this explanation of its nature. It is not necessary that the organism should gain an entrance locally through an abrasion of the mucous membrane; it is more probable indeed that it does so by the blood; for we rarely find evidence of this absorption in cases, such as gastric ulcer or neoplasm, in which an obvious means of ingress is present. It will be remembered that very few cases of phlegmonous gastritis shew any breach of the mucous coat; further, the anatomical characters presented by the openings, if present, suggest that they follow the purulent formations in the submucous coat rather than precede them. To elucidate this point I examined microscopically a number of cases in which the stomach wall was acutely thickened, coexistently with some abrasion of the mucous coat; and I very rarely found any evidence of pyogenetic infiltration. A limited leucocytic invasion of the submucous coat was sometimes seen; but it was noteworthy that the invading cells were chiefly the lymphocytes seen in simple inflammations, and not the multinuclear leucocytes seen in suppurative cases; moreover, organisms were absent. The stomach has a remarkably active power of repair; losses of substance tend to heal by granulation and cicatrisation, and do not readily allow of bacterial absorption. It looks as if pyogenetic absorption of an indirect kind, *i.e.* by way of the blood-stream, may arise in certain cases in which the gastric tissues are sufficiently weakened by any one of several agents, such as alcohol, food-poisoning, starvation, debility. The frequency with which a streptococcus has been found in recent cases, not only in the gastric lesions but in the peritonitis and other complications, is quite remarkable. Accurate bacteriological investigations have been possible in recent cases only, and in practically all of these the streptococcus has been present either alone or in association with the *Bacillus coli*, *B. proteus*,

etc. In some of the cases pure cultures of the streptococcus have been obtained from the gastric lesions, but feeding and other experiments with these cultures have not hitherto been successful in producing the disease in animals. A certain parallel may be drawn between phlegmonous gastritis and the so-called idiopathic cellulo-cutaneous or cutaneous forms of erysipelas. The cause of the latter is the streptococcus so abundantly found in the lymphatic vessels and spaces bounding the affected area; but we do not know how or why it entered. We know that it is more prone to attack patients who are the subjects of general debility, of chronic alcoholism, or of chronic renal or hepatic disease, or patients with an hereditary or acquired disposition; but here our knowledge ceases. Nevertheless, we have not been accustomed to say of the origin of idiopathic erysipelas, as we have been of phlegmonous gastritis, that it is entirely unknown; yet the two diseases have much in common. Indeed it is possible that the same organism, the streptococcus, is the immediate cause of both maladies. It will be remembered that suppuration is usually seen in the more virulent forms of erysipelas. Diffuse phlegmonous gastritis might thus be regarded as an erysipelas of the stomach; Virchow indeed likened the disease to a carbuncle. We are on less certain ground when we attempt to state the conditions which favour the development of the streptococcus in the stomach wall. Alcohol in excess would appear to have a certain influence, perhaps even more than it has in the production of an idiopathic erysipelas, because the stomach, in addition to its increased vulnerability as a part of a deteriorated organism, suffers from the local irritation set up by its contents. Dietetic errors may act in much the same way, excessive loading of the stomach may make it sluggish, and favour the deposition and multiplication of organisms in the submucous coat. Taking all these things into consideration, however, we are compelled to admit that the origin of the disease remains obscure.

Symptoms.—The sudden onset, the epigastric pain, and the vomiting are among the most frequent and trustworthy symptoms; but as peritonitis is such a frequent complication we must endeavour to distinguish the symptoms due to it from those proper to the gastritis. It is present in a large proportion of the cases, probably in one-half. In cases from which it was absent the leading symptoms were epigastric pain, vomiting, fever, restlessness, anxiety, delirium, collapse and coma; and these it will be useful to examine separately.

(i.) Pain was present in about 75 per cent of the cases. In some cases it was preceded by a chill or a rigor. It varied from a mere sense of discomfort or weight in the epigastrium to violent pains of a cutting or boring kind. It generally appeared with more or less suddenness on the first day of the illness; but occasionally not till the second, third, or fourth day. It usually increased in severity. It was mostly continuous, being rarely paroxysmal and rarely subsiding for any length of time. It was nearly always localised at first to the region of the epigastrium, spreading either slowly or suddenly to the rest of the abdomen with the onset of general peritonitis. The early pain is probably due to the irrita-

tion of the nerves so plentifully present in the submucous coat, and to its steady increase on the extension of this irritation and the subsequent implication of the serous surface. The pain is increased by pressure. This is the rule, but in a few cases pressure not only did not increase it, but failed to produce it when previously absent.

(ii.) An increased sense of resistance was generally present also in the epigastric region at an early stage; probably in many cases before the local peritonitis had appeared. Later the upper part of the abdomen frequently became prominent and swollen, and this local meteorism soon became general.

(iii.) Vomiting is nearly always present. Leube says "it was absent in only one of the cases recorded; and even then there was a disposition to emesis." A more extended observation shews that it was absent in about six cases. It was frequently preceded by loss of appetite, irritation, and nausea, generally of short duration, as in most cases the vomiting itself set in on the first day. In one case of five and a half days' duration it appeared on the second day; in another, of eight days' duration, it began on the fourth day; and in another, of nine days' duration, it did not come on till near the end. It was frequently repeated, sometimes almost continuously, without any abatement until death; but in many cases it ceased in a day or two, sometimes altogether; sometimes it came on afresh as peritonitis set in. The vomited matters at first are watery, and contain alimentary matter and mucus; but in almost all cases they soon become tinged with bile, becoming yellow or sometimes greenish. With the exception of one or two cases pus is not recorded as having been present in the vomited matters; and this is remarkable, when we remember that sieve-like openings, large enough to allow of the escape of pus into the stomach, were present in at least five of the cases. Probably its escape into the stomach took place too slowly or in quantities too small to attract attention. It is unlikely that it became altered by the gastric juice, for probably this secretion is largely if not entirely in abeyance. (iv.) Jaundice, generally to a very slight degree, was present in about 16 per cent of the cases. (v.) Temperature.—In most cases there was some fever, the range varying from 100° to 104° F., but in a few cases, in some of which peritonitis was present, there was no perceptible rise of temperature. (vi.) The pulse is generally full, strong, and at first but slightly accelerated. Later it becomes more rapid, feebler, and often irregular. (vii.) The bowels.—Diarrhoea was present in a certain number of cases, constipation in others; sometimes constipation preceded diarrhoea. (viii.) The mouth is generally dry, the tongue is dry and covered with a white fur, and thirst and anorexia are present; hiccup is frequent. (ix.) Mental phenomena.—These varied somewhat, but restlessness and anxiety are generally pronounced from the very beginning of the disease, and increase as it progresses. The patient looks and feels severely ill, and often has the aspect of the "typhoid" condition. Wandering speech and delirium are frequent, and death usually follows from coma or collapse, though the mind may remain clear till the end.

Diagnosis.—The sudden onset, the localised pain increased by pressure, the sense of increased resistance over the region of the stomach, the bilious vomiting and fever, all point to a gastritis. The rapidly increasing severity of the symptoms, the gravity of the case, and the early onset of peritonitis, may suggest its phlegmonous character. Chvostek diagnosed one of his cases during life, and Dörbeck makes the same claim in the case of a woman who suffered from epigastric pain, fever, and vomiting of material containing pus, but recovered, though there was much gastric irritability even seven months afterwards. M'Caskey suggested a diagnosis of phlegmonous gastritis and cancer in a woman who subsequently died and shewed these lesions at necropsy. At least two other observers state that they have made a successful diagnosis during life; but it is obvious that, as a rule, we cannot hope to do more than guess at the diagnosis.

Prognosis.—This is as grave as it can be. The course of the disease is rapid, and its duration brief. The mean duration is about six and a half days. The shortest course was one day, and the longest about seven weeks. A genuine recovery must still be regarded as doubtful. Dörbeck, Lingsmann, Perrin and Blum have observed it, but the diagnosis can hardly be said to have been really established in any of these cases. Dittrich's two preparations in the museum at Erlangen, however, are regarded by Raynaud, Leube, and others as evidence that recovery from interstitial suppuration is possible.

Treatment can be but symptomatic. All alimentation should be by the rectum.

B. **The Circumscribed Form.**—It is not necessary to treat of this form with the same fulness as the former, since we need only discuss the characteristics peculiar to it.

Etiology.—We distinguish between the idiopathic and the metastatic varieties; and the latter occurs probably with a relatively greater frequency than in the diffuse form. Cases are recorded as occurring in the course of puerperal infections, infective endocarditis and like maladies, and in cancer of the stomach. Bacteriological observations are scanty; but following the line of argument employed in discussing the diffuse form, we may fairly conclude that the disease is caused by the arrest, growth, and multiplication of certain pyogenetic cocci within the submucous coat. In all probability it is not invariably due to the same microbe.

Pathological Anatomy.—The whole stomach is not so much altered as in the diffuse form. The abscess is the most prominent change. It varies in size from a bean to thrice the size of a man's closed fist. It may be single or multiple, and it occupies a definitely circumscribed position in the submucous coat. The mucous coat is generally raised inwards over the tumour, and is thinned to a degree which varies with the size of the abscess. It may be pale or reddened, intact, or perforated by one or more apertures. Some authors think that a perforating gastric ulcer may arise in this way. The muscular coat is generally thinned

also, or sometimes softened and destroyed, so that the abscess approaches the serous coat. A purulent peritonitis may thus arise; but it may appear before this and without an actual extension of the pus outwards through the muscular coat. The abscess may rupture into the peritoneal or pleural cavities, into the lumen of the stomach, or even through the anterior abdominal wall. The rest of the stomach may shew little or no change. Secondary abscesses may appear in the liver or other organs.

Symptoms.—These closely resemble those of the diffuse form, especially when the case is acute. The localised pain increased by pressure, and the increased resistance in the epigastric region are, generally speaking, more marked, while the rest of the abdomen is less painful. Premonitory chills or rigors may be noticed. In the chronic cases the symptoms are less pronounced, the pain, vomiting, irregular fever, loss of appetite and strength, and gradual emaciation being the most suggestive. Occasionally all characteristic symptoms were absent. In a few cases a distinct tumour in the epigastrium has been felt during life, which was not always painful on pressure. The vomited matter may contain pus, or pure pus may be vomited in large quantities.

Diagnosis.—In cases in which the symptoms are well marked and a palpable tumour appears in the epigastric region, a diagnosis may be made with some probability. It may become more certain on the vomiting of pus. In Callow's case 20 oz. of pure pus were vomited in the presence of the physician, and other like cases have been recorded. It is obvious, however, that a differential diagnosis between a gastric and perigastric abscess can hardly be made, though it is to be remembered that the latter is considerably more common than the former.

Prognosis is also grave, but several cases of spontaneous recovery after the vomiting of pus have been recorded.

Treatment is symptomatic until the presence of a tumour in the epigastric region renders the diagnosis probable. Surgical interference should then be resorted to without delay. If an abscess be found it should be freely drained. To wait for the possible chance of a natural evacuation of the pus into the cavity of the stomach is dangerous, as every hour increases the risk of a fatal peritonitis. If an operation be undertaken in time, its results ought to be more promising than they are in subphrenic abscess and gastric ulcer, the successful issue of which may now be added to the triumphs of modern abdominal surgery.

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For a fuller account of the disease with a record of cases, see paper by the author in *The Edinburgh Hospital Reports*, vol. iv. 1896. The disease is one of considerable antiquity, as it was noticed by Varandaeus in 1620, *Tractatus de Morbis Ventriculi*. Cases were recorded in the latter half of the seventeenth and the beginning of the eighteenth centuries by Borel, *Opera*, 1656; Sand, *Dissertatio de raro Ventriculi Abscessu*, Regiomont., 1701; Vorwaltner, *Eph. Nat. cur.*, Dec. 3, Obs. 142; Bonetus, *Sepulchretum sive Anatomia Practica*, lib. iii., Geneva, 1700. These all referred to the circumscribed form. The earliest reference to the diffuse form is by Cruveilhier, *vide* Raynaud, *Bull. Soc. anat. Paris*, 1861, tom. vi. pp. 62-93. Among the many papers on the subject, for which see author's monograph above mentioned, are Auvray, *Thèse de Paris*, 1866. Leube, Ziemssen's *Cyclopaedia of the Practice of Medicine*, 1877, vol. vii. p. 154. Fagge, *Trans. Path. Soc. London*, 1874-75, vol. xxvi. p. 81. Chvostek, *Wien. med. Presse*, 1877, Nos. 22, 29, vol. xviii. 693, and *Wien. med. Blät.*, 1881, No. 28, vol. iv. 831, 861, 891, 924, 962. Glax, *Berl. klin. Wchnsch.*, 1879, vol. xvi. 565, and *Deutsche med. Ztg.*, Berl., 1884, No. 3. Ackermann, *Virch. Archiv*, 1869, xlv. 39. Habershon, *Guy's Hosp. Rep.*, London, 1855. Testi, *Ann. univ. di med. e chir.*, Milan, Dec. 1883, pp. 523-547 (with a record of the older and oldest literature). Hun, *New York Med. Journ.*, 1868, viii. 18. Mintz, *Deutsches Archiv f. klin. Med.*, Leipzic, 1892, xlix. 487. Kelynack, *Lancet*, 1896, i. 702.

Membranous Gastritis.—This condition, which is also spoken of as croupous and diphtheritic gastritis, is a rare disease. It is much more frequently met with in children than in adults.

Etiology.—The disease is almost always secondary. A certain amount of evidence has indeed been brought forward to shew that it may be primary, both in children and adults. Niemeyer says that in some cases, in infants, the catarrhal form of inflammation increases to the croupous, but evidence in favour of this is wanting. A few cases have occurred in weakly children as an apparently primary gastritis. A primary occurrence in adults rests mainly upon the case in a man aged forty-six, reported by Delafield.

The secondary form in children may occur at any age. It has been found in new-born children who have died of pyaemia (Bednar, Orth) or of deficient nutrition and debility (Parrot). Later in childhood it most frequently follows diphtheria, as shewn by Jenner, Seitz, Smirnow, Kundrat, Jones, and others. It has also occurred in cases of scarlatina, measles, cholera infantum, and small-pox, in which there was a membrane in the throat also; and a considerable number of cases have been reported in children dying of tuberculosis. In adults the disease is secondary to diphtheria, enteric, typhus, puerperal infection, small-pox, infective endocarditis, corrosive poisons, and so forth. Fox has seen it in phthisis,

and Sir S. Wilks both in gouty kidney and hepatic abscess. It is quite possible that the direct causation varies in different cases. All authorities agree that the condition arises in patients weakened by severe disease, in many of whom there is also a pellicle in the mouth or throat. This pellicle is not always the same. Sometimes it is a simple thrush caused by the *Saccharomyces albicans*, sometimes a true diphtheria, sometimes the membranous exudation of scarlatina. In some cases there may be no distinct membrane, but a haemorrhagic or gangrenous inflammation of the tonsils. The special significance of Löffler's bacillus enables us to distinguish genuine diphtheria from the non-diphtheritic cases, which often simulate it closely. A membranous gastritis may follow upon any such mouth or throat condition when the gastric juices fail to destroy the swallowed germs. Dr. Fenwick states that no free HCl was present in the vomit of a child suffering from diphtheritic gastritis. If the gastric juices retained their digestive power, the false membrane should be digested as quickly as it is formed. The germs of ordinary inflammation, such as staphylococci, streptococci, pneumococci, reach the stomach from the mouth even when there is no mouth or throat lesion, but a membranous gastritis does not follow so long as the gastric juice is secreted. It is in many, if not in most, cases a terminal lesion. The apparently primary cases in children are obscure. They suggest that a simple membranous gastritis may occur in them, like a membranous enteritis or colitis, in which the membrane may be composed of coagulated mucus and a network of fibrin in varying proportions.

Pathological Anatomy.—The mucous surface of the stomach is covered by a membrane of varying thickness, colour, and extent. It is sometimes so thin that it is difficult to keep it whole; at other times it forms a thick and consistent layer. Its colour varies in different cases, and sometimes in different parts of the same case, from a white or whitish-grey to a yellow or even brown or black tint. It usually covers only a small part of the inner surface, and rarely the whole of it from the cardiac to the pyloric end of the organ. It mostly appears in separate patches or elongated strips along the swollen mucous ridges, and usually it is firmly adherent to the mucous membrane beneath, which in turn is usually swollen, reddened, and even haemorrhagic. Generally speaking, it cannot be detached without damage to the surface of the mucous membrane. In a great many cases there is membrane also upon the pharynx or larynx: occasionally it is continuous over the surface of the oesophagus from the mouth to the stomach; but this is rare. Usually the membrane stops abruptly at the junction of the pharynx and oesophagus to reappear in the stomach. Occasionally there is a membranous enteritis or colitis also. The membrane consists of a network of fibrin, containing in its meshes leucocytes, red blood-cells, and degenerated epithelial cells in varying numbers. The fibrin usually forms its main mass, cells of all kinds being few in number, though occasionally it is scanty and the cells, particularly leucocytes, very numerous. The underlying mucous membrane shews a varying degree of change. Degenera-

tive changes in the epithelium, with or without an inflammatory reaction in the deeper tissues, are seen when the false membrane is loosely attached. More extensive changes occur when the membrane becomes more or less firmly attached. The epithelium becomes necrosed. It may lose its structure and, becoming more or less hyaline, blend with the false membrane, as in tonsillar diphtheria. In other cases, though it is necrosed, it retains its anatomical arrangement. The differences indicate different degrees of severity, and it is unnecessary to draw a distinction between a croupous and a diphtheritic form. Bacteriological examination of the false membrane has shewn bacteria to be sometimes present and sometimes absent. Even when present they may be very scanty. Micrococci have been found, and so have bacilli of an indeterminate character. On the other hand staphylococci, streptococci, the *Diplococcus lanceolatus* of Fränkel, the diphtheria and other bacilli, have been recognised in certain cases.

Symptoms.—In many cases there are no gastric symptoms of any kind; in others the ordinary symptoms of severe gastritis are present; namely, vomiting, epigastric pain with or without tenderness, anorexia, thirst, and fever; but in any case the symptoms due to the primary disease are usually so grave as to mask those proper to the gastric condition. The temperature may be normal or subnormal in the apparently primary cases; vomiting, epigastric pain, and a quick pulse being the chief features. Recovery may follow in two or three weeks. Occasionally shreds of the membrane, and even the whole of it forming a cast of the stomach, have been coughed up. It is probably in such cases only that a diagnosis is possible.

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Toxic Gastritis.—This name signifies the changes produced in the stomach by the ingestion of poisonous doses of various substances, of which the chief are concentrated acids and alkalis, arsenic, alcohol, phosphorus, and certain salts, for example, corrosive sublimate, copper salts.

Pathological Anatomy.—The extent and intensity of the changes

depend upon the amount, concentration, and kind of the poison. When dilute, they act only upon the mucous coat; when concentrated, they act on all the coats, producing sloughs more or less deep, surrounded by a zone of intense congestion, and accompanied by a sanguineous effusion into the coats and cavity of the stomach. Alkalis generally penetrate more deeply than acids. The poison acts chiefly upon that part of the stomach wall with which it first comes in contact. Sometimes it acts upon this part only, as peristalsis may be abolished, or the poison be so quickly diluted by other gastric contents that when distributed over the remaining surface it causes no further damage. Thus, the middle of the greater curvature opposite the oesophagus, or the cardiac or pyloric regions, are often chiefly or exclusively affected. Sometimes both the cardiac and pyloric portions suffer, while intervening parts escape. The pylorus is frequently contracted. In the most severe cases the affected part may be completely dissolved, producing a perforation which, though usually circular, varies in size, shews highly congested and haemorrhagically infiltrated margins, and allows the gastric contents to escape into the peritoneal cavity. In the remaining part of the stomach there may be little or no change, although more than half of the organ may have been destroyed. The surrounding organs often shew characteristic changes, or an acute peritonitis may be set up. Sometimes there is no actual perforation, but such complete softening of the stomach wall that it tears upon the slightest touch. In less severe cases the mucous coat is intensely red and injected, often presenting sloughs, false membranes, or ulcers, especially upon the summit of its folds, accompanied by haemorrhagic oedema of the other coats, particularly of the submucosa. The purulent or gangrenous process thus set up may spread for a time, but subside short of perforation, and be counteracted by a healing process. Granulation-tissue is thus formed, which results in cicatrisation; very extensive deficiencies may be partly remedied in this way, as the stomach has a great power of repair. The scar-tissue contracts greatly, and, though the mucous membrane over it may not be regenerated to any extent, the resulting defect may be very small. The contraction, however, may lead to great deformities, such as stenosis of the pylorus, hour-glass contraction, and pouching of the stomach. Baikoff records a case in which the resulting fibrosis reduced the stomach to the size of a watch. This general summary does not take note of the more intimate changes in colour, and other features, produced in the mucous membrane by the several poisons; but a few brief references may be made to them.

The Corrosives.—Sulphuric acid, which is the most common of these poisons, produces a dark red surface with necrosed patches, especially upon the rugae; these are at first grey or greyish-white, later they become dark brown or black, as if charred. This change may be seen over the whole or the greater part of the inner surface of the stomach. The colour will be modified by the amount of food in the stomach at the time, or by the amount of mucus coating its surface. If the coating

be thick the parts appear as if smeared with white paint. Sir S. Wilks mentions a case in a woman who lived several days after drinking dilute sulphuric acid, in which the whole mucous membrane, of a bright yellow colour, lay loose within the stomach. Perforation is more frequent than with any other acid; it occurs in about one-third of the cases. Nitric acid usually produces a yellow or green staining, and less corrosion than sulphuric. The effects of hydrochloric acid sometimes resemble those of nitric, sometimes those of sulphuric acid. No case of perforation has been put on record. Caustic alkalis and their carbonates produce inflammation, with abrasion, ulceration, and haemorrhage. The sloughs are softer, more gelatinous, and less regularly limited. Sir S. Wilks says they convert the mucous membrane into a tawny pulp, and often perforate the stomach. So far as I can find, however, perforation did not occur in any of the recorded cases; but in one case, in a woman who died a few hours after drinking a 30 per cent solution of caustic potash in mistake for iodide of potassium, the stomach was found to be almost completely dissolved.

The Non-Metallic Irritants.—The salts of the alkalis and alkaline earths may produce intense inflammation of the mucous membrane, with ulceration and black patches resembling gangrene. Potassium nitrate and some of the barium salts have been known to cause perforation, but this accident is very rare. Phosphorus causes inflammation and softening of the mucous membrane with frequent petechiae and even erosions. Fatty degeneration of the epithelial cells of the surface and glands quickly follows. Iodine causes inflammation and brown discoloration, sometimes with erosion.

The Metallic Irritants.—Arsenic may cause nothing but a red colour and an injection of the blood-vessels; but after large doses there are usually one or more patches, varying in size from a sixpenny piece to that of a crown, consisting of mucus or of a tough white, yellowish, or even violet-coloured coagulated lymph, mixed with arsenious acid, firmly fixed to the mucous membrane, with signs of intense inflammation around them. White spots of arsenic are also sometimes found between the rugae. The stomach contents are usually of a dark brown colour, sometimes yellow from the partial conversion of the poison into the sulphide. Occasionally the surface of the stomach looks as if it had been coated with yellow paint. Ulceration is rare; perforation and gangrene still rarer. The salts of antimony usually set up intense inflammation only. Corrosive sublimate produces softening, sloughing, and ulceration. The ulcers are often numerous and small, and shew a greyish-white deposit of mercury on their surface. The muscular walls are very soft and friable, particularly in corrosive sublimate poisoning. The salts of copper produce much inflammation and ulceration, and a green or sometimes blue colour. (*Vide* also art. "Metallic and other Forms of Poisoning," Vol. II. Part II. p. 988.)

Organic Irritants.—Oxalic acid acts on the mucous membrane of the stomach, producing a pale or highly inflamed rugose appearance, especially

towards the pylorus. This coat is shrivelled, easily stripped off, and in some cases is partially and even extensively detached; the vessels are minutely injected with black blood. Perforation is rare. Sir S. Wilks mentions a case in which after two weeks a gastritis was set up, and the patient vomited a greenish-brown fluid; after death the mucous membrane was found to be greenish-yellow, injected, and in parts abraded. Alcohol may cause only slight congestion, or an intense dusky red colour and even extravasation. Carbolic acid causes a pale or brown, corrugated, sodden, and partially detached mucous membrane, or a dry surface as if tanned. Cantharides produces intense inflammation and even gangrene, in patches where the powder has adhered. After ethereal oils or acrid vegetable or animal poisons severe injection or membranous inflammation may be seen. Food poisoning may cause intense congestion.

Symptoms.—They are of sudden onset, especially in the case of the corrosives. A sudden penetrating heat is felt in the stomach at the moment when the corrosive reaches it; it soon increases to a vivid burning, excruciating pain in the stomach, oesophagus, throat, and mouth. The epigastrium is sensitive to pressure. There is intense thirst which no drink can assuage; swallowing is painful or impossible; nausea, retching, and vomiting follow, and become more and more frequent, often almost constant. The vomited matters contain food, blood or altered blood, mucus, flakes of epithelium or of the mucous membrane, and frequently traces of the poison. At first the abdomen is usually retracted, but afterwards it may become distended. There is often much bloody diarrhoea coming on shortly after the vomiting; sometimes constipation and tenesmus. The urine is scanty or suppressed. The face is anxious; the pulse frequent, small, and sometimes irregular; the respirations quickened; the extremities cold, and the skin covered with clammy sweat. The lips and mouth are blistered and shrivelled, and may shew spots of a characteristic colour or be cyanosed. The neck and clothes may also shew signs of the poison. In fatal cases the patient becomes cyanosed, his respiration is embarrassed, and he sinks into collapse and dies; it may be by suffocation or in convulsions. All these phenomena come on quickly, sometimes in two hours or even less. The vomiting may cease, but the pain becomes generalised; meteorism and the other classical signs of peritonitis appear, and the patient dies in three or four days. If the quantity swallowed be not very great the symptoms may amend in a few days. The patient may suffer from fever, breathlessness, occasional vomiting, indigestion, and gradual emaciation, and die after the lapse of a few weeks or months. He may vomit portions of the mucous membrane of the gullet or stomach. Laboulbène mentions a case in which, fourteen days after poisoning by sulphuric acid, a membrane was vomited measuring 20 cm. long by 12 cm. broad, in which no glands were found. Rather less than half the adult patients recover completely from the corrosive acids, but recovery may be incomplete, the patient being permanently weakened by means of the cicatricial contraction of oesophagus or stomach or by impairment of digestion. In the case of

such irritants as phosphorus, arsenic, antimony, corrosive sublimate, the copper salts, and the like the symptoms are much the same; but the pain and the vomiting do not usually supervene until some hours have elapsed after the swallowing of poison. Death may follow in twelve to twenty-four hours. If it do not, the symptoms of general intoxication due to the particular poison are soon added to those of the gastritis.

Diagnosis.—This is usually easy. If the patient or his friends do not tell the story, an examination of the mouth will shew whether a corrosive has been taken, and usually also which it is. If the mouth and throat reveal nothing, a careful examination of the vomited matters will usually lead to the detection of the poison, and later the symptoms peculiar to each intoxication may appear, and clear up any remaining difficulty.

Treatment.—The first indication is to get rid of the action of the poison, or to counteract it. In the case of irritants which do not act immediately, the stomach-tube or a free syphonage of the stomach may be promptly used, if we see the case early enough. Emetics, such as powdered ipecacuanha root, mustard, or an injection of apomorphine, may be given when the stomach-tube is inadvisable. In the case of the corrosives its use is contra-indicated, and mild stimulants and diluent drinks should be given, such as white of egg, milk and water—mucilaginous and oily drinks in the case of the acids—with dilute alkaline solutions such as magnesia or chalk and water; or, in the case of the alkalis, with dilute acid solutions such as water and vinegar, or dilute citric acid or lemon juice. These drinks should be frequently repeated for some hours, or even for days. If swallowing be impossible an attempt should be made to introduce a tube beyond the obstruction in the oesophagus, so as to administer the antidote through it. Against phosphorus 40-minim doses of oil of turpentine in mucilage should be given every fifteen minutes for an hour. The oil should be old oil, or French oil (*vide* Vol. II. Part I. p. 997). Magnesia, also in mucilage, should be given next day. Against arsenic the hydrated sesquioxide of iron and the hydrated oxide of magnesia are to be prescribed (*vide* Vol. II. Part I. p. 1077); against corrosive sublimate, the white of egg (*vide* Vol. II. Part I. p. 1008); against carbolic acid, magnesium sulphate (*vide* Vol. II. Part I. p. 1020). The next indication for treatment is to combat the collapse and the pain by injections of ether, caffein, camphorated oil, friction to the skin, injections of morphine, ice or iced drinks by the mouth, and perhaps leeches to the epigastrium. The subsequent gastritis is to be treated in the usual way.

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GANGRENE OF THE STOMACH.—**Pathology.**—A gangrenous condition of part of the stomach wall is seen comparatively seldom. It is most frequently met with in cases of poisoning and when the stomach is the seat of a cancerous growth. Every pathologist is familiar with the foul, ragged, and sloughing masses often seen in this disease; they may be present in the floor and margins of the malignant growth and in its vicinity, and may reach a large size. The cancerous growth, by interference with the vessels, deprives the tissues, especially those of the mucous coat, of their nutrition, and they die. The gastric juice may thus act upon these devitalised tissues as it does upon the cancer itself, and cause their partial or complete solution. It is by this process, more frequently than by the extension of the cancer itself, that peritonitis arises in cases of gastric cancer.

Gangrenous patches or extensive sloughs are frequently seen after the ingestion of various poisons, notably the mineral acids and alkalis. Sloughs of the mucous membrane have also been met with in a few cases of phlegmonous gastritis, and also in membranous gastritis. Occasionally the floor of an ordinary perforating ulcer shews sloughy tissue. It is quite possible that a gangrenous or at any rate a necrotic process may precede the formation of many such ulcers. Necrosis naturally follows the cutting off of the blood-supply to a portion of the stomach wall, and on the solution of this under the action of the gastric juice the ulcer will arise. The microscopic appearances presented by the margins of an ordinary gastric ulcer, not undergoing healing, tend rather to support this statement, for there is none of the vascular or tissue reactions met with in an ordinary ulcer elsewhere. I have met with one case, in a man aged about fifty, in which a gangrenous process was associated with a gastric ulcer. At the lesser curvature, close to the oesophageal end, was an area about the size of a two-shilling piece, partly gangrenous and partly perforated. The perforation was in its upper part; it was about the size of a sixpence, and its upper margin had clearly defined and clean-cut edges; its lower margin was formed by the peritoneal and shreddy subperitoneal coats. This part gradually got thicker, forming a black sloughing tissue which, at the margin of the area, was generally abruptly marked off from the surrounding healthy mucous membrane. An early local peritonitis, most marked in the region of the ulcer, was also present. Mayer's case, reported also by Leube, in which perforation occurred during life near an old cicatrised ulcer, has many resemblances to this case. The perforation occurred in a black, discoloured, irregularly softened area, the characters of which were not unlike those produced by cadaveric softening. It took place suddenly after the patient had partaken heartily of new beer and black bread. Similar perforations have occurred in the oesophagus (Dittrich, Chiari), and it is probable that in all these cases a haemorrhagic infiltration of the walls of the organ had first taken place, and was followed by maceration under the action of the gastric juice. Von Recklinghausen's case of a gangrenous patch about the size of a shilling due to thrombosis of a branch of the

coronary artery, may be regarded as corroborating this view. Habershon describes two cases, one in a woman aged thirty-three with pneumonia and paraplegia, and the other in a man aged fifty-one with pneumonia and renal disease, with several black sloughs situated in cup-shaped depressions in the mucous membrane. Wilson Fox saw sloughs of the mucous membrane in cases of gangrenous pneumonia, and von Recklinghausen describes a case of acute tuberculosis in a man whose stomach shewed several elevated necrotic patches which contained a fungus believed by him to be their cause. Klebs also met with gangrenous patches in the stomach in a case of acute yellow atrophy of the liver in a woman of twenty, and in a case of gangrenous stomatitis in a child of four. Dieulafoy described a case in a man of thirty-five, with abdominal pain, diarrhœa, and vomiting. He had a history of pain in his side for seven days before admission. Two days after admission he had severe hæmatemesis and died two days afterwards. The stomach shewed numbers of erosions with a diameter of 2-3 mm., many partially filled with blood-clot, extending to the submucous coat or deeper, which he ascribed to the pneumococcus which was found not only in the gastric erosions but in a coexistent inflammation of the lungs, of the pericardium, and of the peritoneum. Necessarily rare, however, as such cases undoubtedly are, we may conclude, I think, that severe injury, such as the severe over-distension in Mayer's case, or infective emboli, especially in patients whose strength has been sapped by severe disease, may set up arterial obstruction and consequent infarction, or a direct necrosis. We have, lastly, to determine whether a primary gangrene of the stomach exists apart from the action of corrosives. On hypothetical grounds we may suppose that it does. If primary suppuration exist, why not gangrene? Yet very little evidence can be brought forward in support of this opinion. The sloughing patches which occur in the mucous membrane of the stomach in anthrax can hardly be cited, as they exist only along with intestinal lesions; but Klebs has described a case in which the swollen mucous membrane of the stomach shewed a few brownish-red patches like partially gangrenous areas. These presented an abundant round-celled exudation, and numerous spore-containing bacilli, varying in length from 5·9 to 11·7 μ; these were found upon the free surface and in the lumen of the gastric glands, frequently separating the cells from the basement membrane. There were no round cells except where the bacilli were present, the latter apparently coming first. In the light of more recent work it is doubtful if these bacilli were not merely accidental. Nasse and Orth describe a somewhat similar case in a man aged sixty who suddenly fell ill and died after vomiting much blood. The diagnosis was gastric ulcer, but the necropsy shewed many surface losses in the mucous membrane, and that the gangrenous process had spread deeper than in the last case, having at one place eroded a branch of the coronary artery. Bacilli were present in these lesions also. It would thus appear that a local primary gangrene of the

stomach may occur, but that it is among the rarest of pathological curiosities.

Symptoms.—There are no symptoms proper to the gangrenous process, except the appearance of shreds of the mucous membrane in the vomited matter; and this happens chiefly in toxic gastritis. They should be carefully looked for in other cases, however, as I was once able from a microscopic examination of a shred of tissue thus brought up to diagnose a cancer of the stomach in a man under thirty years of age, in whom all the other symptoms were consistent with simple catarrh or ulcer. In most cases the gangrene is entirely overshadowed by the concomitant disease, and in others the signs suggest no more than an ulcer or an acute gastritis.

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Cirrhosis of the Stomach.—In this, one of the rarest of stomach affections, there is marked thickening of the submucosa, accompanied also by thickening in the muscular and serous coats. It has been described by various writers under different synonyms, namely, plastic linitis by Brinton, hypertrophy by Wilson Fox, fibroid induration by Handfield Jones, sclerosis by Sneller, cirrhosis, and so on.

Etiology.—The number of cases recorded, though small, has rapidly increased of recent years. They are almost equally divided between the two sexes, and the average age of occurrence in man seems to be about 50, and in woman somewhat under 40. Observers generally seem to think that the most important point in the disease is the determination as to whether it is cancerous or not. All the earlier writers (Andral, Cruveilhier, Brinton, Habershon, Wilson Fox, Wilks) have denied its cancerous nature, though it must be admitted that the microscopic part of the evidence upon which they relied was imperfect. They considered the disease as a primary cirrhosis of the submucosa of a simple inflammatory nature, homologous with the common cirrhosis of the liver; and regarded the changes seen in the other coats as subsequent and subsidiary thereto. Bret and Paviot have sought to prove that the lesion is really cancerous; they base their contention, not so much upon the microscopic appearances of the submucosa, which they admit even in their own cases did not shew a structure clearly indicative of cancer, as upon the condition of the perigastric lymphatic glands—which did shew characteristic epithelial cells—and upon the presence of

metastatic growths. They argue, further, that all previously reported cases, from their strong similarity to their own, were really of the same nature. This is a doubtful inference. They have succeeded in shewing only, what was long believed, that a cirrhotic atrophic cancerous infiltration of the walls of the stomach may so simulate a simple cirrhosis of that organ as to be indistinguishable from it, save by a most careful and searching microscopic examination. Hoche, Vautrin, Cignozzi, Rivèt, and others have recorded cases which they state to be cancerous in nature, and it is not improbable that several of the cases recorded as simple may have been cancerous. In my own case the wide extent of the lesion made cancer seem improbable, and microscopic examination shewed beyond all doubt that this impression was correct. There is nothing inconsistent or improbable in the supposition of a simple gastric cirrhosis. We meet with a like condition in other organs, as, for example, in the liver and kidney; and in the deeper layers of the skin beneath the epithelium we find dense fibrous growths, either localised or diffuse, in cheloid and sclerodermia respectively, for the origin of which we are unable to account. It would thus appear that a cirrhotic condition of the stomach may arise either from a simple or a cancerous condition. In the latter case, when no metastatic growths are formed, the cancer becomes atrophic and the condition becomes practically identical with cirrhosis of non-cancerous origin. That is, the cancer as such does not appear to have much influence; it is the gastric cirrhosis which follows which is of chief importance. Alcohol was regarded by Brinton as a likely exciting cause, but little dependence can be placed upon it, for not only is the disease rare as compared with the frequency of over-indulgence in alcohol, but we find that in the great majority of cases there is no history of alcoholism. Syphilis is shut out in a similar manner. An attempt has been made to shew that the disease spreads from without, and chronic perigastric peritonitis is pointed to as the prior condition, and as the cause of the stomach changes. It is, however, quite as probable that the changes are concomitant, or even that in some cases the sequence is reversed. There is another and quite distinct form of cirrhosis which frequently follows chronic gastric catarrh and atrophy of the glands. Here the first change is in the mucous coat, which becomes thinned, atrophied, and sometimes sclerosed. The inflammation extends to the submucosa, generally in an irregular way, giving rise to patches of fibrous thickening, sometimes of considerable size, which are most frequent and abundant in the region of the pylorus, and may lead to a certain amount of hypertrophy of the muscular coat. The cavity of the stomach is generally dilated. In a well-marked case under the care of Prof. Clifford Allbutt the process seemed to have arisen in the cicatrices of healed ulcers, whence it invaded the whole stomach. The organ was much contracted, and the patient ultimately died of pyloric stenosis. The ulcers—simple ulcers which healed soundly—occurred between the ages of twenty and thirty, and death ensued from stenosis some ten years later. Judging from the

scars the ulcers had been of considerable extent and severity, and the result a sort of cheloid.

Pathological Anatomy.—The stomach as a whole is markedly diminished in size, firm, rounded, and elastic. In advanced cases it resembles a thick-walled india-rubber tube, blown out at one end (cardiac). It is increased in weight, and when opened does not collapse. When cut it often gives rise to a creaking sound like cartilage, and there is considerable resistance to the passage of the knife through its walls. The cavity is much reduced; in some cases its capacity falls to little more than two ounces of fluid. The thickening of the wall is greatest in the pyloric region, frequently attaining six to eight times the normal mean, and it may even reach the thickness of an inch. It gradually diminishes until the cardiac extremity is reached, where it may be slight or practically absent. In a certain number of cases it implicates the pyloric half only of the organ; very rarely, indeed, does it affect the whole organ uniformly.

It is said not to pass beyond the pyloric and oesophageal openings respectively; but in a case of my own this change was found throughout the gastro-intestinal tract from stomach to rectum, excepting the duodenum and upper jejunum. The aperture of the pylorus may or may not be contracted. The colour of the section of the stomach wall is a dull opaque white, and all the coats—mucous, submucous, muscular, and serous—stand out clearly from one another. The chief thickening is in the submucosa; but it may be surpassed by that of the muscular coat. The mucous membrane is usually pale and smooth, or ridged or mammillated, that is, beset with little projections closely studded together; but it is sometimes congested, sometimes excoriated and ulcerated. The microscope usually shews very little thickening, atrophy, or signs of catarrh. The gastric tubules are rarely separated, bent, or deformed by any interstitial fibrous growth. The cells may be granular or fatty, but in many cases, and in many sites in one case, they may seem very little altered. In my own case there was in places a fairly abundant, small round-celled exudation, of a recent nature, irregularly distributed between the tubules; and the secreting cells themselves were smaller than normal, and often detached. The submucosa is firm, opaque, white or greyish, and glistening. Microscopically it is seen to be composed of white fibrous tissue arranged in irregular bands and networks. The fibres are rarely dense, but are usually well formed and developed, and only occasionally rudimentary and mixed up with many cells. Very little notice has been taken of the condition of the blood-vessels. In my own case they were comparatively few in number, and shewed thickening of their outer coats and sheaths, but no signs of any endarteritis. In a number of cases cancer-cells have been found, while in a few the embryonic nature of the growth has been suggestive of sarcoma. In my own case there was no resemblance whatever to either the one or the other. The muscular coat is thicker, firmer, and paler than normal; it is penetrated by fine bands of fibrous tissue, visible even to the naked eye, passing outwards from the submucosa to the subserosa. Under the

microscope the muscular fibres are seen to be greatly increased in numbers, especially in the circular layer; and generally they shew also an infiltration of fibrous tissue, either restricted to definite strands, or diffusely and intimately mixed up with the muscle-fibres, as, for example, in an old myoma uteri. The serous coat with its subserosa is generally thickened, often considerably, so that its surface has a dull white opaque look instead of its normal clear and shining appearance.

Other signs of extensive or subacute peritonitis are not wanting in many cases; for thickenings and opacities of the gastro-hepatic and gastro-colic omenta, of the tissues behind the stomach, and other tissues around, or of the peritoneum elsewhere, have been observed. Exudation of lymph on the intestinal coils, fibrous adhesions, ascites, general dropsy, or cirrhosis of the liver may coexist with it.

Symptoms.—It would appear that in some cases the disease has run its course without giving rise to any symptoms, death being due to some intercurrent malady. In such cases the changes are said to be slight. In the great majority of the cases well-marked symptoms have come on, sometimes suddenly, sometimes insidiously. Pain in the epigastrium is the chief of these. It varies greatly in different cases, and at different times in the same case. It may be slight or very severe. It comes on at first only after food, generally immediately after, and usually becomes more severe as the case progresses. Vomiting is also very frequently present; it sets in after the pain, at first occasionally—say, three times a week or so—but gradually increasing in frequency and severity, until it may occur many times a day. The vomited matters may consist of the ingested food, of clear watery mucus, or later in the disease also of coffee-ground material, or even pure blood. The haematemesis, which is due to the congestion and sometimes to the ulceration of the mucous coat, is occasionally copious. Sometimes there is tenderness on pressure over the epigastric region; but this symptom is often delayed. The appetite is bad, though it may remain good for a considerable time, and the patient may complain that formerly he could take a good meal and now is troubled with a sense of distension or pain or vomiting before his hunger is satisfied. There is usually constipation, at any rate for a considerable time. There is at the same time a steadily advancing anaemia and emaciation. Since these are symptoms mostly characteristic of chronic gastric catarrh and of perforating ulcer, it is obviously impossible by their means to diagnose between them; but as the gastric cirrhosis advances a tumour becomes palpable in the epigastric region: occasionally below the ribs on the left side, and exceptionally on the right side: it varies in size, is round or sausage-shaped, firm and smooth, is fairly movable from side to side, and also slightly from above downwards. If caught with the hand and prevented from moving when deep respirations are taken, it is said to slip immediately upwards when liberated. Percussion over the tumour is never dull, and generally gives a resonant or moderately resonant note. An ascites has been found in some cases, and a general dropsy in others. Cirrhosis of the liver is

occasionally present also. The anaemia and emaciation advance steadily, though for a long time the blood count may be comparatively high, and the vital powers gradually diminish ; the patient usually dies from asthenia. The rapidity of the process seems to vary greatly ; it generally lasts for many years, though in some cases the symptoms date back for only a few months or even less. It is probable that the disease is then fairly well established before it gives rise to any symptoms. In Prof. Allbutt's case the symptoms were those here described, but no dropsy appeared. Not only could the stomach be manipulated and its size ascertained, but its movements were very active, and conspicuous in both directions.

Diagnosis.—The appearance of the tumour is the first trustworthy indication of the nature of the disease ; it enables us to set aside simple gastric catarrh and ulcer. The presence of a tumour is equally characteristic of cancer ; but from this disease we may be able to distinguish it by the smoothness, mobility, and semi-tympanitic note of the tumour, the time of onset of the vomiting and the pain, the infrequency of haematemesis, the absence of signs of metastasis, and the long duration of the malady. Prof. Osler and others have in this way made successful diagnoses. Peristalsis may be visible. Free hydrochloric acid is absent from the gastric juice in many cases whether cancerous or not. The stomach may be washed out and an attempt made to ascertain its capacity; if it does not hold more than a few ounces the diagnosis will be supported.

Treatment.—Under the ordinary treatment of gastric catarrh or of ulcer the earlier symptoms often improve greatly for a time. Later, when the nature of the disease becomes probable, the chief indication is to support the patient's strength. A mild and bland nutritious diet should be given in small quantities, and repeated at more frequent intervals than usual. Milk, combined at times with cream, strong soups, beef essences, raw minced beef, and so forth, should be chiefly relied upon. Nutrient enemas may also be used. Repeated lavage of the stomach with dilute soda solution has been tried in some cases with a favourable result. The quantity of fluid introduced should, it is said, be increased gradually until the patient can bear about one litre with comfort. Among medicines opium is the most highly recommended. Cocaine internally, and leeches, mustard blisters, or hot applications to the epigastrium, may also be tried for the purpose of alleviating the pain. Gastric tonics may do some good. Cases of recovery are reported by Schnetter and also by J. W. F. Smith ; but in estimating their value the difficulties in the way of an indubitable diagnosis during life have to be considered. Roux records a case in a man of 33 on whom he performed an anterior gastro-enterostomy to relieve the distressing symptoms. The stomach wall was greatly thickened and its cavity greatly diminished. The operation was difficult owing to the rigidity and friability of the gastric walls, but the patient made a good recovery and was in good health 3½ years afterwards.

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ULCER OF THE STOMACH

By the late Prof. Julius Dreschfeld, M.D., F.R.C.P.

Synonyms.—*Perforating ulcer*, *Simple ulcer*, *Round ulcer*, *Peptic ulcer.*

Definition.—An affection of the stomach characterised clinically by localised pain, vomiting, and haematemesis; and pathologically by a circumscribed loss of substance of one or more coats of the stomach by a process which appears to be a necrobiosis.

Etiology.—*Frequency.*—Gastric ulcer may be regarded as fairly common, but its precise incidence is very difficult to formulate. Statistics based on post-mortem records are, as Dr. Byrom Bramwell has pointed out, apt to be fallacious, inasmuch as all open ulcers are not discovered, cicatrices of healed ulcers are often overlooked, superficial ulcers may heal without leaving visible scars; and lastly, some cicatrices may be due to causes other than gastric ulcer, such as syphilitic and tuberculous lesions. Neither can clinical statistics be relied upon to give a fair conception of the frequency of the disease, as cases which on accurate investigation prove to be hyperchlorhydria, gastralgia, and so forth, are in many instances returned as ulcer. Dr. Hale White even believes that the usually accepted statistics of ulcer are rendered entirely fallacious, more especially with regard to the age and sex incidence, by the failure

to differentiate cases of haematemesis which depend upon an oozing of blood from the gastric mucosa ("Gastrostaxis," *vide* p. 479).

The incidence of gastric ulcer, as shewn in the post-mortem and clinical records available, exhibits some variation in different parts of the world. Post-mortem statistics give a frequency varying from 1 to 20 per cent of all autopsies; Welch found open ulcers or scars in 5 per cent of 32,052 autopsies collected from continental sources, which is identical with the percentage determined by Brinton. In Berlin gastric ulcer appears to be relatively infrequent; Berthold, from observations between the years 1868 and 1872, noted its occurrence in 2·7 per cent of the autopsies at the Charité. In Munich it was observed by Nolte in 1·2 per cent of 3500 autopsies. In Zürich Stahl observed it in 2·16 per cent. In Erlangen it was noted by v. Ziemssen in 4·55 per cent. In Kiel, by Griess, in 8·3 per cent. In Copenhagen gastric ulcer is more common, being found in 13 per cent by Starcke, and in 20 per cent by Grünfeld. In London the Fenwicks noted the occurrence of gastric ulcer in 4·6 per cent of 10,000 autopsies. In America Campbell Howard (75) tabulated open ulcers or scars in 1·32 per cent of 10,841 autopsies performed in ten hospitals of seven cities. Among the first thousand autopsies at the Johns Hopkins Hospital there were nine examples of ulcer of the stomach (Osler).

The frequency of gastric ulcer as shewn in clinical statistics varies as follows:—In Breslau and Zürich Lebert found gastric ulcer in 0·66 per cent of 41,688 medical admissions between 1853 and 1873. In the Charité Hospital of Berlin ulcer was met with in 1·33 per cent of 42,219 admissions between 1888 and 1898. In London the Fenwicks noted the occurrence of ulcer in 0·82 per cent of 45,712 admissions to the London Hospital and the London Temperance Hospital in ten years. In 5000 consecutive outpatients it was met with in 0·74 per cent; the frequency for inpatients and outpatients combined being 0·78 per cent. In Edinburgh gastric ulcer appears to be more frequent; in 35,692 medical admissions to the Edinburgh Royal Infirmary in eight years it occurred in 2·2 per cent. In 7665 outpatients it was met with in 0·09 per cent. The combined inpatient and outpatient incidence being 2·02 per cent (Byrom Bramwell), almost three times that of London. In America Campbell Howard found that 0·57 per cent of 161,599 admissions to the medical wards of fifteen hospitals distributed among eight cities were diagnosed as gastric ulcer.

Sex.—Gastric ulcer is more frequent in females than in males. Here again the data obtained from post-mortem and clinical statistics do not agree; and further, there are variations in different localities and at different periods. The generally accepted ratio, as determined from autopsy statistics, is 2 to 1 (Brinton, Danziger). At the Manchester Royal Infirmary the incidence in females was somewhat greater, 2·5 to 1; other observers give a somewhat lower rate of incidence, as 11 to 8 (Steiner), 1·7 to 1 (Habershon (69)), 134 to 126 (Riegel), 3 to 2 (Welch, the Fenwicks). In Breslau Lebert found it to be more common in males—

3 or 4 to 1—while in Zürich it was more common in females. Clinical records shew a still higher incidence in females, 3 to 1 (the Fenwicks), 3·5 to 1 (Lebert), which would seem to indicate that whilst females are the more susceptible, they are less liable to succumb to the disease. The Fenwicks, distinguishing between acute ulcers which heal readily and except by perforation rarely prove fatal, and chronic ulcers which are progressive, heal tardily and often imperfectly, find in 89 autopsies with open ulcer, 30 acute, 3 in males and 27 in females; and 59 chronic, 43 in males and 16 in females. From this they conclude that whilst acute ulcers are almost confined to young females, the chronic form of the disease is more common in males.

The disparity in the post-mortem and clinical statistics of the sex incidence of gastric ulcer is difficult to explain; it may be due to the scars of acute ulcers being more easily overlooked, or to the inclusion in the clinical conception of gastric ulcer of some other condition which is relatively very common in females, as has been advocated for some years by Dr. Hale White.

Age.—Generally speaking, gastric ulcer occurs most frequently between the ages of 20 and 40—in females between 20 and 30, in males between 30 and 50. Brinton's statement that the frequency of gastric-ulcer appears to increase, allowing for the number of persons living at the various age periods, with advancing years, being based on the occurrence of ulcers and scars in cadavers of various ages, is open to the fatal objection that scars, being ineffaceable records of a previous lesion, will apparently increase with each decade, but will afford no evidence of the age of incidence of the lesion itself. In 171 cases collected from the records of University College Hospital, in which the age-incidence was compared with the duration of the symptoms, 15 cases occurred between the ages of 0 and 20; 75 between the ages of 20 and 30; 38 between 30 and 40; 25 between 40 and 50; 14 between 50 and 60; and 4 in persons over 60 (S. Martin). In 383 clinical cases the Fenwicks found the age-incidence as marked by the first symptom to be very different in the two sexes—in males 30 per cent occurred between the ages of 10 and 30; 59 per cent between 30 and 50; and 11 per cent over 50 years: in females 75 per cent occurred between the ages of 10 and 30; 21 per cent between 30 and 50; and 4 per cent over 50 years. Before puberty gastric ulcer is very rare; yet it has been observed in infants and occasionally soon after birth. The melaena of the newly-born, though often due to erosions and small multiple ulcerations, has in some cases been found to be due to simple ulcers of the stomach with regular, well-defined borders, varying in diameter from 1 to 3 centimetres, and situated in the stomach or duodenum (42). According to Kundrat haemorrhagic erosions are very frequent in children, and he believes they differ from the true ulcer of the stomach in degree only. Dr. Goodhart reported a case of ulcer of the stomach in an infant 30 hours old. According to Prof. Osler, ulcers of the stomach have been found in the foetus and in the new-born shortly after birth. Martha Wollstein found five cases of

gastric ulcer out of 390 autopsies at the Babies' Hospital in New York (95). The Fenwicks have collected 18 cases in infancy and childhood, of which 13 were acute and 5 chronic, the ratio of females to males being 4 to 1. After 70 years of age death from gastric ulcer is also rare, but that it may occur and make itself manifest is shewn by such cases as that of Weber, who records a fatal haematemesis in a female aged 72; of Sedgwick, who noted perforation in a male aged 82; of Fenwick, over 80 years; of Eppinger, who records a case of a man whose age was reputed to be 120 years. I have seen several cases of gastric ulcer in subjects over 70 years of age, its existence in one being verified by operation.

Race and Climate.—From statistical records the frequency of the disease appears to vary in different places. Thus, it appears to be more common in Europe than in America (Dacosta, Welch, Howard); in Europe it is more frequent in the North than in the South, and less frequent in France than in the adjacent countries. In Britain it appears to be much more common in Edinburgh than in London. In America it is more common in the north-eastern than in the more southerly regions, with the exception of San Francisco. In Eastern Siberia Sperk states that it is very common, whilst according to von Sohlern it is very rare in some districts of Russia and in the Bavarian Alps. This relative infrequency is attributed by von Sohlern to the diet, which consists largely of vegetables, and thus leads to the presence of a large proportion of potassium salts in the blood. Westphalen denies that hyperacidity and ulcer are rare in those districts where but little meat is eaten. He found hypersecretion in from 25 to 30 per cent of all cases of gastric disease among his Russian patients; and in the district of Oberessen, where the diet is composed of vegetables almost exclusively, both ulcer and hyperacidity are common.

Occupation.—According to Bamberger gastric ulcer occurs more frequently in cooks, and he attributes this to their habit of tasting food when it is very hot. From our own hospital statistics it appears to be more common in housemaids, but as these are generally anaemic and of the age when gastric ulcer is most frequent, it is not improbable that age and sex are more significant than occupation. Eichhorst states that it occurs frequently in metal-turners; and he thinks it is produced in them by the irritation of particles of dust which are swallowed. Bouveret has noted its frequent occurrence in mirror-polishers, and Bernutz is of opinion that porcelain-workers are liable to the disease for a similar reason. Occupations which necessitate direct mechanical pressure on the epigastrium, such as those of tailors, shoemakers, weavers, and the wearing of tight belts or corsets, are said to exert a disposing influence in the causation of ulcer (Rasmussen).

Hygiene and Habits.—It is said to be more common amongst the poorer classes, who take insufficient or badly prepared food, and live in badly ventilated, small rooms. If this be true, it is probably only in so far as anaemia is produced by these various factors. The opinion of

Lancereaux that it is more common in drunkards, especially in those who take ardent spirits, is not confirmed by our own observations, nor by the age and sex incidence of the disease.

Associated Diseases.—Gastric ulcer is said to be more frequently observed with certain diseases; notably chlorosis, amenorrhoea, tuberculosis, syphilis, malaria, scurvy, chronic valvular affections of the heart, chronic Bright's disease, septicaemia and pyaemia. Chlorosis is very constantly associated with gastric ulcer, as the primary disorder. The Fenwicks found chlorosis in 72 per cent of their cases of acute ulcer. It is generally regarded as well established that gastric ulcer is prone to occur in young chlorotic females; it is also equally certain that a certain degree of secondary anaemia may follow repeated haemorrhages from the stomach. The significance of this association and the particular changes met with in the blood will be dealt with in the sections on Pathogenesis and Symptomatology respectively.

Menstruation.—The frequent association of menstrual disorders and gastric ulcer in females has been emphasised by some writers to the extent of distinguishing a special form of the disease under the term "menstrual ulcer" (Crisp). In the Fenwicks' cases of acute ulcer in young females, in whom chlorosis almost invariably coexisted, amenorrhoea was noted as preceding the ulcer in 19 per cent, as following it in 11 per cent, and in 16 per cent the menstrual function was either deficient or irregular, while in 54 per cent it was normal. The anomalies of menstruation are, however, to be regarded rather as phenomena of the blood condition than as factors in the production of the ulcer. The influence of menstruation on the functions of the stomach cannot be gainsayed, and in cases of gastric ulcer an exacerbation of the symptoms is occasionally noted during these periods, as in a case under the observation of Dr. Craven Moore, in which with each menstrual period there was haematemesis and an excess of pain and hyperaesthesia. Von Jaksch believed gastric ulcer to be exceptionally frequent after parturition, but it has not been possible to verify this opinion.

Tuberculosis.—The occurrence of pulmonary tuberculosis in about 17 per cent of all cases of gastric ulcer has led to the belief that the pulmonary disease might dispose to the gastric lesion. On the other hand, an open ulcer of the stomach was found by Dittrich in only 1 per cent of fatal cases of pulmonary tuberculosis, and at the Brompton Hospital in 0·9 per cent. It has been suggested that if any relation at all obtain between the two lesions, it is in the other direction, that the ulcer of the stomach may serve as the portal of infection by the tubercle bacillus, in support of which several somewhat dubious cases are recorded; or, and this seems more probable, that the gastric lesion may dispose to pulmonary infection by inducing a condition of malnutrition and lowered resistance. On the other hand, specific ulceration of the stomach, due to the tubercle bacillus, is occasionally met with, and may be difficult to differentiate clinically from the simple ulcer. The earlier pathologists regarded tuberculous ulceration of the stomach as extremely rare, this

viscus being rendered almost immune by the presence of hydrochloric acid and the paucity of lymphoid tissue. But already quite a considerable number of such cases have been recorded (71, 150). Tuberculous ulcer of the stomach is generally multiple, it is rounded in form, the edges are raised, thickened, smooth or worm-eaten, and undermined, and the base is generally irregular. As a rule the gastric lesions are associated with similar changes in the intestine and peritoneum, and with extensive tuberculosis of the lungs.

Syphilis.—Some observers regard syphilis as an important factor in the production of gastric ulcer. Lang states that 20 per cent of all cases of gastric ulcer occur in syphilitic subjects; others place the ratio at 10 per cent, but this is certainly much higher than in my own cases. Dieulafoy (36) believes that when signs of simple ulcer of the stomach appear in a syphilitic subject it may be presumed that the lesion is specific, and that antisyphilitic treatment is indicated. It may be assumed that an ulcer of the stomach can be of syphilitic origin in one of the following ways:—(1) By the softening and breaking down of a gumma in the wall of the stomach; such a contingency must be very exceptional, as gummas in this situation are exceedingly rare, even in congenital syphilis (46, 56). The resulting ulcer differs from the simple gastric ulcer in not having clean, punched-out edges with a smooth base, but in appearing as a deep excavation in the submucous tissue, with irregular or overhanging edges, and a base that is covered by a greyish slough or cheesy irregularities. Further, the adjacent mucous membrane not infrequently contains numerous small gummas embedded in its substance. (2) Syphilitic endarteritis and obliteration of the gastric vessels may induce necrosis or a diminution in the resisting powers of the stomach wall. This sequence has been demonstrated by Luxenburg and Zawadski, but it is most exceptional. (3) As a consequence of syphilitic anaemia (cachexia) acting in the manner to be presently discussed. In certain cases of ulcer of the stomach which had resisted the ordinary methods of treatment, it was found that they rapidly yielded to antisyphilitic medication (122). The validity of using this as proof of the specific nature of the gastric lesion is, however, open to question.

Chronic valvular disease of the heart is not uncommonly associated with gastric ulceration, usually as multiple small ulcers chiefly about the fundus, and arising apparently in small erosions, due to passive engorgement and haemorrhage. Less frequently a large ulcer of the indolent or chronic type is met with which may possibly have had a similar origin.

Chronic Bright's Disease.—Haemorrhagic erosions and indolent ulcers of the stomach are—the former frequently, the latter occasionally—associated with chronic nephritis and passive engorgement. In addition the stomach may present lesions similar to those met with in the intestine in this disease, described as uraemic ulcers (99), but these probably have no significance in the causation of the simple ulcer of the stomach (see also p. 574).

In septicaemia and pyaemia, acute pneumonia, anthrax, enteric fever, erysipelas, general tuberculosis, and in all conditions inducing severe

degrees of passive congestion of the gastric mucosa, it is not unusual to find numerous small excavations in the mucous membrane chiefly in the region of the fundus, which are possibly the result of embolism of the gastric vessels, or of capillary haemorrhages into the deeper layers of the mucosa. The etiological relations of these so-called erosions will be dealt with subsequently (p. 458). Ebstein records an instance of the association of gastric ulcer with *trichinosis*; in a young girl who had eaten large quantities of trichinous meat five round ulcers were found in the pyloric region.

Traumatism.—It seems very probable that under certain conditions simple external injury, such as blows, kicks, falls, or continuous mechanical pressure on the epigastrium and abdomen, may produce simple ulcers of the stomach. A blow on the stomach or sudden severe pressure on the epigastrium may be followed by profuse haematemesis, and later by epigastric pain and vomiting of food for some weeks, but these symptoms, as I have on several occasions observed, rapidly disappear under treatment. Occasionally, however, the condition produced by the injury is more resistant, and the clinical manifestations become those of chronic ulcer of the stomach; instances of these effects have been recorded by many observers (Potain, Bouveret, Derouet, Leube (91), Wagner, Limont and Page, Duplay). Ackermann observed in Cohnheim's clinic 16 cases of post-traumatic ulcer of the stomach; in 15 continuous pressure or repeated severe pressure on the abdomen, necessitated by occupation, and in one a kick on the abdomen appeared to be the exciting cause. Fertig records an interesting case of traumatic ulcer: a stableman received a kick on the epigastrium, his stomach being full at the time. Laparotomy was performed, but the external aspect of the stomach and intestine was normal. Five days later haematemesis and melaena occurred, and death ensued the following night. At the autopsy there were four recent ulcers on the lesser curvature. Ebstein (44) mentions two cases of gastric ulcer produced by the strain of lifting heavy weights, which appears to have induced rupture of some gastric vessel and a haemorrhagic infiltration. Recently Dr. Craven Moore had under his care a robust middle-aged man, who strained himself whilst pushing a heavy waggon, and at once vomited a large quantity of blood; this haematemesis was subsequently repeated, and proved fatal a week later; at the autopsy a recent circular ulcer was found about the centre of the posterior wall. There was no other lesion of note in the body. During life there was neither epigastric pain, tenderness, nor hyperaesthesia. A curious example is described by Pauly (2) in a man who, to avoid a fall, threw himself violently backwards; immediately following this he began to suffer from slight gastric disturbance, and six weeks later he succumbed to perforation of a recent ulcer. Richardière, who records a number of cases of gastric ulcer of traumatic origin, divides them into two classes: one characterised by very violent initial symptoms, which rapidly subside and disappear under treatment, and the other by a more protracted course, resembling that of a characteristic ulcer of the stomach. This difference,

he believes, depends on variations in the gastric juice; with normal acidity healing rapidly ensues; in the presence of hyperacidity it goes on to the gradual development of a characteristic gastric ulcer. The case is different when the traumatic cause is an extensive burn of the skin. Though when symptoms of ulcer follow a burn, the seat of it is almost always in the duodenum (*vide* p. 557), yet in a few cases the ulcer has been found in the stomach. Swallowing corrosive poisons has in a few instances been followed by the formation of a gastric ulcer (57).

Heredity.—Most authors are silent on this subject; a few who mention hereditary influence in the causation of gastric ulcer look upon the evidence as unsatisfactory. I have taken some pains to inquire into this matter. In a few cases the account of gastric ulcer in various members of the same family rested on insufficient grounds, but in eight cases the evidence appeared conclusive; in two instances mother and daughter suffered from it; in one, father and daughter; in two, two sisters; and in one instance brother and sister suffered from unmistakable symptoms of the disease. The Fenwicks noted the occurrence of the disease in one of the parents in about 3 per cent, and in some other member of the family in nearly 5 per cent of their cases. In almost every instance the disease was of the acute form, and affected the young female members of the family.

Pathological Anatomy.—Ulcers having the characteristic appearance of gastric ulcer are only found in the stomach, duodenum, the lowest part of the oesophagus, and in the jejunum after gastro-enterostomy (p. 574).

Number.—Usually one ulcer only is found; occasionally two or even more may be found; if two are found, they are sometimes opposite to each other; more often they are close together, one generally being chronic or even cicatrised, the other of recent date. In 463 cases Brinton noted 2 ulcers in 57; 3 in 16; 4 in 3; 5 in 2; and more than 5 in 4 cases. The Fenwicks in 867 cases of open ulcer found the percentage of single and multiple ulcers to be as follows:—One ulcer in 80·5; 2 ulcers in 12·1; 3 ulcers in 3·1; 4 or more ulcers in 4·26; so that the lesion appears to be multiple in about one-fifth of all cases. Among 112 cases observed at the London Hospital a distinction was drawn between the acute and chronic varieties, and it was found that whilst the acute was multiple in 54 per cent, the chronic variety was multiple in only 13 per cent. Occasionally a large number of ulcers have been found—34 by Berthold; and Lange speaks of a case in which they were too numerous to count.

Situation.—Most commonly the ulcer is found at the pylorus, more frequently on the posterior wall near the lesser curvature; occasionally it is on the anterior wall (more often in acute than in chronic ulceration); in rare instances the ulcer is on the greater curvature, and more rarely still on the fundus of the stomach, these parts *in toto* being affected in only one-fifth of all cases. The following tabulation of 1015 cases and the comparison with the statistics of Welch is from the Fenwicks' monograph:—

	Fenwicks.	Welch.
	Per cent.	Per cent.
Pylorus	15·6	12·0
Lesser curvature	36·0	36·3
Posterior surface	25·0	29·3
Cardia	7·9	6·3
Greater curvature	4·1	3·4
Anterior surface	8·0	8·7
Fundus	3·3	3·7

In 70 cases of chronic ulcer the distribution corresponded with the above, whilst in 39 cases of acute ulcer the distribution was—pyloric portion, 13; middle portion, 14; cardiac portion, 12; and in 26 the ulcer was nearer the lesser curvature, and more frequent on the posterior surface. The acute ulcer, therefore, has not the same proclivity for the pylorus as the chronic ulcer has. The ulcers so frequently situated about the lesser curvature are only occasionally found exactly on the line of the curvature itself. With regard to ulcers in the vicinity of the two orifices, it is interesting to note that the cardiac orifice itself is more often the site of such an ulcer than the pyloric opening.

Size.—The size of the ulcer varies. Generally it is a little less than an inch in diameter; but the diameter may be smaller, less than half an inch, or larger, measuring two inches or more. Small ulcers are usually acute ulcers. In rare instances, as in the cases recorded by Cruveilhier ($6\frac{1}{2} \times 3\frac{1}{2}$ in.) and by Law (6×3 in.), ulcers much larger still have been found. In the case of a woman who died at the Manchester Infirmary, in a small stomach measuring about five inches from the cardiac end to the pylorus and only two inches in circumference at the broadest part—a stomach smaller than the transverse colon—an extensive cicatrix was found extending from the cardiac end to the pylorus; there was a history of profuse haematemesis fifteen years before death, but during her stay in the hospital the patient had no dyspeptic symptoms and could take the ordinary amount of food.

Appearance.—This varies according to the duration of the disease, and we may distinguish between *acute* ulcers of recent origin and *chronic* ulcers. The acute ulcer is usually round, occasionally oval; its characteristic appearance is as if it were punched out of the wall of the stomach; the edges are clean cut, smooth, not thickened; the floor is smooth, occasionally irregular, firm, or somewhat pulpy; in rare cases it may be covered by a slough of necrotic tissue. Small haemorrhages and congestion may occasionally be found on the edges and in the neighbourhood of the ulcer. The depth of the ulcer varies; it may extend to the submucous, muscular, or even serous coats, and as each successive layer of the wall of the stomach may be less destroyed than the preceding layer, the ulcer may present a funnel-shaped appearance, or be like a terrace with sloping edges, and not uncommonly its axis is directed obliquely, follow-

ing the direction of some branch of the coronary artery. The explanation of this characteristic form is usually that it corresponds to an infarcted area [Virchow]; but it also may be due to the different contractility of the several coats of the stomach, as shewn by Dr. Soltau Fenwick, who produced a funnel-shaped lesion by perforating the stomach wall with a cautery or a thread of uniform diameter. In ulcers of long standing the appearance is altered by the occurrence of inflammatory changes in its edge and base; the ulcer is generally larger, round, oval, irregular, or occasionally, if of very long standing, girdle-shaped or horseshoe-shaped with signs of scarring. The edges are thickened, irregular, or may be slightly undermined; the funnel-shaped appearance may be lost or even better marked than in the recent or acute form of ulcer. The floor of the ulcer is either smooth or irregular, and is formed by more or less dense cicatricial tissue. The surrounding mucosa often shews chronic inflammation and very frequently a zone of congested and varicose vessels. The serous surface both in the acute and chronic forms of ulcer very frequently shews a fibrinous deposit, localised to the base of the ulcer without either perforation or adhesion. This, as pointed out by Mr. Mayo Robson, is an important surgical guide, and its frequency has been shewn to be 74·3 per cent (97). Occasionally the serous coat and the base of the ulcer are so thickened by chronic inflammation as to form a definite tumour; in several such cases which have come under my observation it proved difficult, even when the stomach was exposed at operation, to decide as to the nature of the mass, although its benign character was proved by the favourable issue of the cases.

Histologically the acute ulcers do not shew any changes other than destructive, either at the borders or in their floor; in the chronic ulcers, however, there are signs of inflammation. The glands at the border of the ulcer appear enlarged and tortuous, some are changed into small cysts, in others the epithelium has undergone a marked change; we can no longer distinguish between the central and the parietal cells; the whole lumen is distended with small cubical and cylindrical cells, which also line the walls of the glands; the interglandular tissue is thickened and contains newly formed fibrous tissue; cell-infiltration and new formation of fibrous tissue are also found in the submucous and muscular layers. The floor of the ulcer presents similar appearances; the muscular layers are more or less completely replaced by fibrous tissue which, in ulcers of long standing, forms dense fibrous layers. The arteries about the base of the ulcer often shew mes- and endarteritis and thrombosis, and occasionally well-marked hyaline changes (Openchowski).

Apart from these changes it would appear from the observations of Galliard and of Jaworski and Korczynski that the mucous membrane of the stomach in all cases of gastric ulcer shews changes which are more evident on microscopic examination. These changes consist in a cell-infiltration between the several layers of the coats of the stomach and marked inflammatory changes in the walls of the blood-vessels (both of veins and arteries), and in the neighbourhood of the nerves also. Jaworski

and Korczynski examined portions of the walls of the stomach removed at operation in cases of gastric ulcer, and found very similar changes, together with the degeneration and disintegration of the central cells. These changes were found in sections taken from various parts of the mucous membrane of the stomach, even when a considerable distance away from the disease. According to these authors the inflammatory changes are constant and primary in gastric ulcer; but many pathologists look upon them as secondary, and as not always present.

Adhesions.—The inflammation of the serous coat over the base of the ulcer may lead to the formation of adhesions, at first fibrinous, later fibrous, to adjacent viscera. The frequency of adhesions in chronic ulcers is estimated by von Jaksch (77) at 40 per cent, by Lebert at 42 per cent, and by the Fenwicks at 46 per cent, whereas in acute ulcers the latter noted adhesions in 2 cases only out of 33. The relative frequency with which the various adjacent viscera are implicated is shewn in the following analysis of 123 cases by the Fenwicks:—

Pancreas alone	in 40 per cent		
Liver alone	in 26·8 „	74·8 per cent.	
Pancreas and liver	in 8·1 „		
Colon	in 5·7 „		
Liver and colon	in 3·2 „		
Spleen	in 1·6 „		
Mesentery	in 2·4 „		
Three or more organs	in 12·2 „		

Thus, as the ulcer is most often on the posterior wall of the stomach near the pylorus, the stomach is most commonly (75 per cent) adherent to the pancreas and the left lobe of the liver. Ulcers near the cardiac end of the greater curvature may contract adhesions to the spleen, omentum, diaphragm, and so on. In the course of time ulceration may extend through the fibrous adhesions, and the floor of the ulcer may then be formed by the adherent organ, such as the pancreas denuded of its peritoneal surface, the liver, or the spleen. In one case I found that the floor of the ulcer was of an irregular and convex outline, and consisted of necrotic liver tissue, and on further examination that the ulcerative inflammation had eaten away a considerable mass of the adjacent liver tissue. The spleen is rarely affected, but cases of fatal haemorrhage from erosion of the splenic pulp have been recorded. If the ulcer be on the anterior wall of the stomach, there are, as a result of the greater mobility of the part of the stomach affected and of the absence of any fixed organ in the neighbourhood, either no adhesions or very slight ones; hence it is that ulcers situated in this locality so often perforate.

In some cases the inflammation of the serous coat is progressive and may eventually implicate large areas. Such diffuse perigastritis, more commonly found in connexion with ulcers about the cardia, may lead to considerable matting of the stomach and adjacent viscera—liver, spleen, diaphragm, abdominal wall, pancreas, and colon—and to considerable

deformity and diminution in the size of the stomach. More rarely peritoneal bands or false ligaments may originate in such secondary inflammation, and Robinson and the Fenwicks describe cases in which such a band has constricted the stomach into a bilocular sac with chronic ulceration of the mucosa along the line of pressure.

Perforation of the ulcer may occur in any part of the stomach, if the adhesions be not very firm. Brinton first pointed out that perforation is specially apt to occur in ulcers on the anterior wall; this, no doubt, depends on the extreme mobility of this part of the stomach and the absence of any fixed organ in its vicinity to which adhesions might be formed. He found 70 per cent of all perforations on the anterior wall, 21 per cent on the lesser curvature, and 9 per cent on the posterior wall. The Fenwicks' analysis of 351 cases shews that a difference obtains in the frequency with which different parts of the stomach are perforated in acute and chronic ulcers.

		Anterior.	Posterior.	Pylorus.	Middle.	Cardia.	Lesser Curvature.
		Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.
Acute .	Operation	80·5	19·5	20	24	56	50
Acute and Chronic	Post-mortem	62	38	45	30	25	44
Chronic .	Post-mortem	36	64	62	26	12	60

It thus appears that whilst the acute ulcer usually perforates the anterior wall, near the lesser curvature, and in the cardiac portion of the stomach, the chronic ulcer is more prone to perforate in the pyloric portion, on the posterior wall, and near the lesser curvature.

The results of the perforation vary according to the size of the perforating opening and its situation. If the ulcer perforate into the peritoneal cavity, and if the opening be large, some of the contents of the stomach may pass into the peritoneal cavity and give rise to perforative peritonitis; if the opening be very minute, there may be but a local peritonitis and a formation of fresh adhesions by which the small opening may be occluded; if perforation take place into an adjacent portion of the intestine, a bimucous fistula is established; such a fistulous communication is most commonly formed between the stomach and transverse colon, occasionally between the stomach and duodenum, or between the stomach and another portion of the small intestine; much rarer are fistulous tracts between the stomach and the pericardium, heart, pleura, gall-bladder, or a bronchus. The inflammation set up does not always lead to fibrosis, it may result in the formation of an *abscess*, in 10 per cent of fatal cases (Lebert). In most cases, no doubt, the abscess is the result of a small perforation, as, for instance, the subphrenic abscess, or abscess between the stomach and the anterior abdominal wall;

whether such an abscess may form without previous perforation is difficult to prove (*vide* art. "Subphrenic Abscess").

Haemorrhage.—Gastric ulcer being a form of necrosis, we can readily understand how in the course of the destructive process blood-vessels become eroded, and haemorrhage, of varying degree, occurs. This is as frequent in the acute as in the chronic ulcer. Microscopically, the arteries often shew periarteritis and endarteritis. This change, when the artery is small, leads to more or less diminution of the lumen of the vessel, and may end in the formation of a thrombus by which haemorrhage is prevented. In other cases the walls of the small arteries are softened and transformed into embryonic tissue which may cause or aid the haemorrhage so often observed in ulcers (33).

If a haemorrhage be fatal we may often detect the open mouth of a fairly large artery in the centre of the floor of the ulcer. The microscopic examination of the vessel in one case shewed scarcely any pathological change in the walls of the artery itself, except at the ruptured end, where a mass of granular detritus was found. In such cases, and when capillary vessels only are eroded, the haemorrhage may be too slight to be manifest to the naked eye in the vomit or in the fæces [occult haemorrhage of Boas (10)]. The more severe degrees of haemorrhage result from erosion of a vessel, artery or vein, in the submucous or subserous coat, or from invasion of some neighbouring vascular organ, as the liver, spleen, or heart. The vessels most liable to be the source of a profuse haemorrhage are the coronary, the epiploic arteries and their branches, and the splenic artery. In 48 per cent of Brinton's cases of fatal haemorrhage the ulcer was on the lesser curvature, in 34 per cent on the posterior surface, and in 4 per cent on the anterior surface. In 74 such cases the Fenwicks found the ulcer on the lesser curvature in 54 per cent, on the greater curvature in 6 per cent, and on the posterior wall in 40 per cent. In 66 cases in which the source of haemorrhage could be accurately determined, a branch of the coronary artery was eroded in 32, the splenic in 14, the right gastro-epiploic in 6, the gastro-duodenal in 4, the splenic vein in 3, the coronary veins in 3, the left gastro-epiploic artery in 2, and the plexus of veins around the cardia in 2. Perforation of the spleen with profuse haemorrhage (v. Jaksch, Steiner), of the liver, pancreas, adrenals, and of the heart (4 cases), are occasional accidents.

Cicatrisation.—The acute and the chronic ulcer alike naturally tend to undergo complete cicatrisation, as is shewn by the statistics of the occurrence of scars in autopsies; thus in Copenhagen, where gastric ulcer appears to be particularly prevalent, Grünfeld found scars in 20 per cent of all autopsies. If the ulcer has been superficial, not penetrating to the peritoneal coat, we notice only a slight depression in the mucous surface, often concealed by adjacent rugae, and slight thinning of the wall of the stomach; if, however, the ulcer has penetrated to some depth, the cicatrix appears as a dense white scar of stellate form, slightly depressed and surrounded by radiating folds of thickened mucosa, and is often

associated with a distinct puckering of the serous coat. The contraction of the scar may give rise to some alteration in the size and shape of the stomach and so prove a source of further trouble.

Pyloric Stenosis and Dilatation of the Stomach.—The most frequent sequel to cicatrisation is stenosis or narrowing of the pylorus with subsequent dilatation of the stomach. This obtains in from 16 to 20 per cent of all chronic ulcers in the vicinity of the pylorus, but in about 2 per cent only of these is the stenosis extreme. In a few cases the pyloric stenosis appears to be due not to cicatricial contraction but to spasm induced by an ulcer in its immediate vicinity (pyloro-spasm); or it may be due to deformity of the pylorus or to adhesions fixing the pylorus to the under surface of the liver and subsequent kinking at the pyloro-duodenal junction.

Contraction of the Stomach.—This condition may arise from a diffuse perigastritis, from constriction of the cardiac orifice by cicatrisation of a chronic ulcer in its vicinity (2 cases out of 112 (Fenwick), 1 out of 39 (Starcke)), from spasm of the cardia, or as in the case related on p. 450, from the cicatrisation of a very extensive ulcer.

Irregular Deformity.—When the ulcer is on the lesser curvature, its healing may lead to the dragging together of the two orifices of the stomach, which may then become adherent by fibrous tissue.

Hour-glass Deformity—Bilocular Stomach.—A considerable number of examples of this comparatively rare condition have been recorded as due to the cicatricial contraction of a healing ulcer. Usually the ulcer is on the lesser curvature and has extended transversely to the long axis of the stomach, or there may have been two ulcers, one on either side of the lesser curvature. The stomach appears, by the resulting constriction, to be divided into two pouches, the larger corresponding to the fundus, the smaller to the pyloric portion of the organ, the sulcus separating the two is generally of considerable depth and situated somewhat nearer the pylorus than the cardia. Posteriorly the stomach is often adherent to the pancreas and to the under surface of the liver. Internally, the cardiac pouch is smooth and its wall is thinned; the pyloric pouch often shews signs of inflammation and its wall is thickened. Occasionally, however (Robson and Moynihan), the pyloric pouch is also dilated, the result of a coincident cicatricial or spastic stenosis of the pylorus. The communication between the two pouches varies in diameter from that of a pencil to such as will admit one or more fingers. More rarely cases are met with in which two constrictions divide the stomach into three pouches. Two examples of this "trifid stomach" are recorded by Mr. Robson and Mr. Moynihan.

Diverticula.—Localised bulging of the stomach wall in the situation of an ulcer is a curious and rare deformity; 3 cases have been described by Kleine, 2 others by Grassenberger and Kolaczek, and one by Dr. Craven Moore. In all, the ulcer was situated near the pylorus and was associated with pyloric stenosis. Kleine accordingly regards the deformity as analogous to pulsion diverticula in other situations.

Pathogenesis.—Our knowledge of the exact causation of gastric ulcer is as yet far from definite, and many views are held as to its pathogenesis; this is not surprising, as the ulcer is not always due to one and the same cause; in many cases, indeed, several causes operate together, some more immediate, some more remote. From a consideration of the morbid anatomy two points stand out clearly; the first that in the acute or recent ulcer, inflammatory changes are conspicuously absent; the other that the lesion is confined to those parts of the alimentary canal which are subject to the action of the acid gastric juice, the stomach, the lower end of the oesophagus, and the duodenum as far as the biliary papilla (*vide* also peptic ulcer of jejunum, p. 574). On the basis of these it may be premised that the essential factor in the pathogenesis of the lesion is one which acts locally on a circumscribed area of tissue in such a way as to diminish its resisting powers, and renders it vulnerable to the eroding activity of the gastric juice. The latter, inasmuch as its digestive influence must be equally distributed over the entire mucous membrane, and not localised in a circumscribed area, can only be a contributory factor; and even anomalies in its composition can endow it with no greater significance. The nature of defective tissue resistance is obscure, and many views have been propounded to explain this elusive point—a loss of some vital principle inherent in the cells of the mucosa (Hunter); a depression of the proliferative powers of the deeper cells of the mucosa on which normally the restitution of the surface layers depends, as these are being continuously destroyed by the digestive action of the gastric juice (Claude Bernard); a cessation in the circulation of the alkaline blood-plasma (Pavy); necrosis or necrobiosis of the tissues brought about in various ways—disturbances of circulation, trauma, bacteria and toxins, inflammation; a solution in the continuity of the protecting mucus-clad surface layer of epithelium (Bloch, Schmidt); a neurotrophic change (Wilks, Stockton); a disappearance of specific protective substances or anti-ferments from the cells of the mucosa (Weinland, Cooke). But whatever be the precise nature of the change or series of changes rendering the tissues vulnerable to the gastric juice, it has been shewn by direct experiment on animals that it may be induced in circumscribed areas in many different ways and result in the formation of a lesion, which, whilst resembling in structural peculiarities the round ulcer in the human stomach, differs from it in that it almost invariably heals promptly and shews no tendency to extend. Thus, the problem of the pathogenesis of the gastric ulcer is less concerned with the production of the ulcer than with the elucidation of those conditions which interfere with its repair and at the same time give it a progressive character. The explanation of this characteristic of ulcer of the stomach has been sought for in alterations in the composition of the gastric juice, in the composition of the blood, in the vital activity of the tissues generally, and in certain structural peculiarities in the stomach wall. The several factors responsible for the circumscribed loss of tissue, and for its assumption of a progressive character, will now be considered in detail.

A. *Factors which may destroy the Immunity of the Living Tissues to the Digestive Action of Gastric Juice in Circumscribed Areas of the Stomach.*—1. Circulatory Disturbances.—Virchow, recognising the close analogy between ulcer of the stomach and infarcts in other organs in respect of size, shape, and vascular relations, regarded the lesion as a localised ischaemic necrosis induced by embolic occlusion of some branch of the coronary artery. Virchow's opinion obtained wide acceptance and exerted a profound influence on subsequent observations, being confirmed experimentally by Panum and Cohnheim. This simple explanation of the pathogenesis of gastric ulcer will, however, suffice for a few cases only. The occurrence of embolism of the gastric vessels in infective endocarditis where other organs are extensively affected is quite exceptional, and experimentally it has been shewn that of emboli thrown into the circulation comparatively few reach the stomach and duodenum, and those that do are generally situated in the region of the fundus. Again, it is very exceptional to find in the class of subjects in whom gastric ulcer is chiefly met with, any correlated lesion from which emboli might originate or any evidence of the embolic process in other organs. Embolism of the gastric arteries, when it does occur, is usually secondary to disease of the large vessels in the neighbourhood, as aortic atheroma, aneurysm of the coeliac axis, or in the course of various septicaemias, the emboli consisting of bacteria, as in pyaemia, anthrax, or, as shewn by the experiments of Klebs and Welti, of disintegrated red blood-corpuscles, a possibility which has been advanced to explain the occasional occurrence of gastric ulcer in malaria and in some severe anaemias (haemolytic). The possibility that circumscribed necrosis may be secondary to arterial thrombosis in an artery is also remote, for thrombosis occurs chiefly in arteries with diseased walls; gastric ulcer, on the other hand, occurs much more frequently in young persons in whom degenerative and other changes in the walls of the blood-vessels are very uncommon. On the other hand, it is not improbable that thrombosis following atheroma of the coronary arteries may be an important factor in the production of gastric ulcers in middle-aged subjects, or in younger subjects when it supervenes on syphilitic endarteritis (Luxenburg) or on lardaceous disease of the vessels in pulmonary tuberculosis (Merkel, Edinger). Klebs suggested that a local anaemia of the gastric mucosa, and therefore gastric ulcer, might be induced by temporary spasm of the gastric arterioles: this is purely hypothetical and has little to support it, apart from some experiments made by Talma, who found that stimulation of the left vagus produced tonic spasm of the pylorus and, by the compression of the contained arterial trunks, ischaemia and ulceration in the pyloric portion of the stomach.

Venous Stasis and Haemorrhage.—It has been shewn by Müller and Dr. Fenwick that sudden occlusion of the portal vein results in intense venous engorgement of the gastric veins and haemorrhages into the mucosa which frequently proceed to ulcerations in the central and cardiac regions of the stomach. These lesions differ markedly from the ordinary gastric

ulcer, and further, as shewn by Dr. Fenwick, obstruction to individual branches of the gastric veins is unattended by any deleterious consequences. Rindfleisch and Axel Key believed that venous obstruction, haemorrhages, and consecutive ulceration might result from compression of veins by spasm of the pylorus.

Haemorrhagic Erosions.—This term is applied to small blood-stained abrasions of the mucous membrane of the stomach which are a fairly frequent post-mortem phenomenon (1·8 per cent of all autopsies, Willigk) in new-born children, in various cachectic conditions, in chronic heart disease, obstructive pulmonary disease, in hepatic cirrhosis, and various septicaemias, pneumococcal, pyogenetic, tuberculous. As a rule the erosions are multiple and chiefly in the fundus of the stomach, individually they are circular in form, from 2 to 4 mm. in diameter, and in depth extend half way through the thickness of the mucosa (simple erosions of Dieulafoy) or down to the submucosa (exulceratio simplex of Dieulafoy). The base of the erosion is generally blood-stained and the tissues in that situation infiltrated with blood. They apparently originate in one of two ways, by capillary haemorrhage following stasis or from small haemorrhagic infarcts following capillary embolism. Some observers have regarded these erosions as a possible starting-point of gastric ulcer, but the consensus of opinion is against this view, and is supported by their number, situation, absence of transitional phases, and their different age- and sex-incidence. Clinically they may be of no importance, or they may give rise to characteristic manifestations (see p. 479).

II. Trauma.—(*a*) Mechanical.—Clinical observation has shewn that gastric ulcer may follow on various mechanical insults to the stomach. Experimentally Ritter found in dogs that severe blows on the epigastrium, the stomach being distended with food, produced haemorrhage into the submucous coat (submucous haematoma), which he surmises would have proceeded to an open ulcer by the eroding action of the gastric juice. These results were confirmed by Vanni in experiments on rabbits. More recently Gross has conducted a series of experiments with similar results, and he concludes that in otherwise normal stomachs injuries to the mucosa would not lead to ulcer unless very severe. Direct injury of the mucous membrane, as shewn by the experiments of Quincke and of Griffini and Vassale, does not give rise to the formation of ulcers; this may often be verified when on washing out the stomach large pieces of the mucous membrane are accidentally detached, for the defects caused by such injuries heal very readily. A. Schmidt has called attention to the contraction of the stomach wall, by which means any lesion of the mucous membrane is covered over and shut off from the cavity of the viscus. He believes that traumatic lesions which are not obliterated in this way will probably develop into an ulcer provided there is active gastric juice present. But the great majority of such traumatic lesions promptly heal in the absence of other factors (see p. 448). Schmidt attributes the predilection of ulcer for the lesser curvature and the pylorus to the mucosa in this part of the stomach being less mobile than elsewhere. Continued

pressure, such as produced by tight lacing or tight belts, may be an occasional cause. Rasmussen pointed out a distinct sulcus in the anterior wall of the stomach which passes vertically from the small to the large curvature close to the pylorus, and is opposite the lower line of ribs; this line of depression, which shews thickening of the serous coat, atrophy of the mucous surface, and occasionally peritonitic adhesions, he looks upon as due to pressure; and in several cases he found scars of gastric ulcer along this line.

(*b*) Chemical.—We can readily understand how such ulcers might be due to poisoning with corrosive substances, whether acids or alkalis; but this, after all, will explain very few cases.

(*c*) Thermal.—It has been thought that scalding food might produce a limited amount of necrosis, and in this way the apparently frequent occurrence of gastric ulcers in cooks has been explained. Decker's experimental investigations support this, for by introducing very hot food by means of a stomach-tube into the stomachs of two dogs, he produced in one case marked haemorrhagic erosion, and in the other a characteristic acute ulcer of the stomach. In a few cases gastric ulcer in man may be produced in this way, but certainly only in a few; for slight loss of substance of the stomach wall heals readily, and even if a haemorrhagic erosion should follow, it by no means always gives rise to gastric ulcer (Langerhans).

III. Neuropathic Disturbances.—Secretion, vascularity, and other functions of the stomach being dependent on many nervous influences, the nervous system is held by some authors to play an important part in the causation of a gastric ulcer. It may do so in various ways. We know that after injuries of the nervous system, such as excision of the corpora quadrigemina (Ebstein, Brown-Séquard), intersection of the thalami and peduncles (Schiff), hemi-section of the medulla (Schiff), section of the spinal cord (Schiff, Koch, Ewald), injection of absolute alcohol into the coeliac plexus and into the splanchnics (Dalla Vedova), and sometimes also in diseases of the central nervous system, punctiform haemorrhages, or haemorrhagic erosions are found in the stomach. But, even if vasomotor disturbances, leading to the small haemorrhages, were more common, it is very doubtful whether small haemorrhages or haemorrhagic erosions lead to gastric ulcers. By section of the vagus below the diaphragm Yzeran was able to produce in 9 out of 21 rabbits allowed to live for more than two weeks ulcers of the stomach, generally single and located in the pyloric region. He believes the mechanism of their formation to be spasm of the pylorus followed by interstitial haemorrhage and erosion by the gastric juice. Somewhat similar results were obtained by Saitta. Talma's experiments, in which ulceration was induced by faradic stimulation of the left vagus in rabbits, have been alluded to. More recently Donati has repeated Vedova and Yzeran's experiments on dogs and rabbits with negative results except that in some increased acidity of the gastric juice was induced. A similar effect was observed by Dalla Vedova as a result of his experiments. The significance of this hyper-

chlorhydria in the genesis of gastric ulcer will be discussed below (p. 461), but it may be at once premised that there is abundant evidence that an increased secretion of hydrochloric acid in the stomach may be readily brought about by nervous influences. The importance of the nervous system in the pathogenesis of gastric ulcer was somewhat differently regarded by Wilks and Moxon, who looked upon gastric ulcer as a trophic lesion analogous to the so-called trophic ulcer of the cornea. Stockton believes that some distinct and persevering nerve-perturbation is the basis of the defective resisting powers of the tissues which renders them vulnerable to the gastric juice.

IV. Gastritis.—The early writers on gastric ulcer, such as Abercrombie and Cruveilhier, looked upon chronic catarrh of the walls of the stomach as the primary cause of gastric ulcer; this view was abandoned after the vascular infarct hypothesis was proposed by Virchow and Rokitansky, but it has been revived by Galliard and by Jaworski and Korczynski, who found signs of gastritis in different portions of the stomach in cases of chronic ulcer. Dr. Soltau Fenwick believes that under certain conditions inflammation of the solitary lymphoid follicles of the stomach may give rise to an acute ulcer. These small accumulations of lymphoid tissue lie in the mucosa superficial to the muscularis mucosae; they are present in greatest numbers up to the age of puberty, and from then onwards gradually diminish in number and size, disappearing from the cardia towards the pylorus and lesser curvature, so that in these latter situations they are always most abundant. In gastritis and various infective disorders such as enteric fever, acute tuberculosis, diphtheria, pneumonia, these follicles become acutely inflamed, their tissue undergoes rapid necrosis, and numerous small pits or erosions with congested edges are found in the gastric mucosa. It would appear highly probable that many, if not all, of the so-called haemorrhagic erosions already described originate in inflammation of the lymphoid follicles, especially when these occur in young subjects. In many cases they apparently have but little significance, but on the other hand they may give rise to serious haemorrhage from erosion of the subjacent capillaries, and are very possibly the pathological basis of some cases of gastric haemorrhage in young females (see p. 479). Recently, Dr. C. H. Miller, who from the examination of a large number of human stomachs has confirmed Dr. Fenwick's observations, also regards inflammatory disintegration of these follicles as a starting-point of gastric ulcer when, from the coexistence of chronic inflammatory changes in the tissues of the stomach wall, local contraction is unable to take place and the base of the erosion remains exposed to the action of the gastric juice.

V. Bacterial Infection.—Boettcher found micrococci in the borders and floors of gastric ulcers. Letulle more recently expressed the opinion that some of the gastric ulcers may owe their origin to microbes. Gastric ulcers have been noticed in pyaemia, puerperal septicaemia, and other infectious diseases; more often, however, instead of typical gastric ulcers, haemorrhagic erosions have been found, and microbes have been detected

in them. Again, injection of pure cultivation of microbes, such as the *Staphylococcus pyogenes*, into the stomach or into the peritoneum gives rise to haemorrhagic erosions, and even to ulcerations. The relation of ulcerations in the stomach and intestinal tract to micro-organisms has recently been studied more closely, and it has been clearly demonstrated that ulcerations of the alimentary tract in animals may be caused by the invasion of various micro-organisms into the deeper layers of the mucous membrane and the lymphatic glands connected with it, where they set up necrosis. This fact has been made use of by Dr. Sidney Martin to explain the formation of chronic gastric ulcer by bacterial necrosis; and the more frequent occurrence of gastric ulcer at the pyloric end he explains by the absence here of the glands secreting hydrochloric acid, which has a powerful antiseptic action and prevents the invasion and growth of bacteria. Stokes records a case of gastric ulcer shewing the diphtheria bacillus, which he regarded as the causal agent. Turck asserts that he has produced characteristic ulcers of the stomach in dogs by the administration by the mouth of emulsions of the *Bacillus coli communis*. But as yet this view, so far as the gastric ulcer of man is concerned, may be looked upon as purely hypothetical.

It will be seen from the above that there are many factors which are capable of interfering with the vitality of the stomach wall, and in a circumscribed area, in such a manner as to render it vulnerable to the eroding activity of the gastric juice. In most instances, however, in which the power of one or other of these factors can be tested experimentally it is found that the resultant lesion either never attains the form of the stomach ulcer or that it heals with great rapidity. Accordingly, the co-operation of other influences must be taken into account in the complete formation and progressive character of the gastric ulcer.

B. *Factors which lead to the characteristic Form of the Initial Lesion.*—(1) Local Conditions in the Stomach.—Experimentally it has been shewn that lesions in the vicinity of the lesser curvature and pylorus heal more tardily than those situated nearer the fundus. This has been explained by the pyloric portion of the stomach being the seat of most vigorous movement, lesions in this situation accordingly getting but little rest; and by Schmidt it is looked upon as being due to the relatively slight mobility and slight redundancy of the mucosa in this part of the stomach, in consequence of which defects in its substance are obliterated with greater difficulty. The similar significance of chronic inflammatory changes interfering with the local contractility especially of the muscularis mucosae has been emphasised by some observers.

(2) The Gastric Juice.—In addition to its action in removing the devitalised tissue, the gastric juice has been regarded as a potent factor in the further progress of the ulcer and in the prevention of its healing, by virtue of its containing free hydrochloric acid. The importance of an excess of free hydrochloric acid in the gastric contents in the production of a gastric ulcer has been emphasised by Riegel, who maintained that hyperacidity was a feature of all cases of gastric ulcer and was primary

to that lesion. Matthes and Schmidt shewed experimentally that whilst traumatic lesions of the gastric mucosa invariably failed to develop into ulcers, this would occur if coincident with the infliction of the injury the stomach was filled with 5 per cent hydrochloric acid. Similarly Koch and Ewald found that the erosions usually following section of the cervical cord would become ulcers if 5 per cent hydrochloric acid were introduced into the stomach. On the other hand many observers look upon hyperacidity of the gastric juice as inconstant, and when present as secondary to the ulcer of the stomach (Pawlow); thus, whilst Riegel regarded hyperacidity as constant, having found it in a series of 31 cases, others have found it much less frequently, as is indicated below:—

	Ewald (49) (132 cases).	Wirsing (116 cases).	Howard (76) (54 cases).
	Per cent.	Per cent.	Per cent.
Sub-acidity . .	9·0	1·8	26·4
Normal acidity .	56·8	55·5	56·0
Hyperacidity .	34·1	42·7	17·6

Occasionally cases are met with in which free hydrochloric acid is persistently absent (Howard, Agéron).

(3) Alterations in the Blood.—The precise relation between an ulcer of the stomach and chlorosis with which it is so frequently associated is uncertain. It has been suggested that the abnormal state of the blood might by leading to thrombosis or capillary haemorrhage be a primary cause. On the other hand the chlorosis might interfere with the healing of a lesion of the gastric mucosa and belong solely to the second category. Experimental evidence supporting this last supposition has been provided by Quincke and Daettwyler, Silbermann, Fütterer, and Cohnheim, who have shewn that artificial lesions of the gastric mucosa in animals rendered anaemic heal more tardily than in normal animals, whilst the production of artificial anaemia has not lead to ulcer. It would, therefore, appear that though anaemia rarely initiates the lesion in the stomach, it is an important factor in its elaboration by interfering with its repair: the explanation of this may lie in some general depression of the reactive powers of the tissues or, as suggested by Riegel, on some associated alteration in the composition of the gastric secretion, for it has been shewn by Schätzell, Grüne, Cantu, and others that hyperchlorhydria is present in from 73 per cent to 95 per cent of all cases of chlorosis.

Symptomatology.—The symptoms vary considerably; in some cases the ulcer may be entirely latent, or give rise to no characteristic signs; in others, especially the acute ulcers, the first marked symptom may be profuse haematemesis or the symptoms of acute perforation. In most cases, however, besides dyspeptic disturbances, there are certain symptoms which may be looked upon as characteristic; namely, localised pain in

the epigastrium coming on at a variable period after food, vomiting and haematemesis. In long-standing cases, apart from these stomach symptoms, we often see signs of anaemia and of malnutrition.

If we consider the symptoms separately, we have to note:

Pain.—This is perhaps the most frequent symptom, being present in from 80 to 90 per cent of all cases. The patient complains of pain situated mostly in the epigastrium, just below the ensiform cartilage; it is distinctly local, and usually covers but a small area; occasionally it starts at the epigastrium, and extends thence to the left or to the right; in very chronic cases the pain is often situated some distance below the epigastrium, and is more diffuse; in most cases, besides the epigastric pain, there is also pain in the back, either over the region of the spine between the sixth and eighth dorsal vertebrae, or to the left of the spine and then situated somewhat lower, between the tenth dorsal and first lumbar vertebrae. The pain varies in character and intensity; usually it is of a heavy boring character, at other times it is described as a burning pain, in some cases as a shooting pain, and at times it may become much more intense, more like a colicky pain (gastralgia), and may lead to syncope; in the early stage the pain is usually much less severe. The pain is not continuous, it comes on after meals, sometimes very shortly after food is taken, and in this case probably the irritation by the solid food is the cause of it; at other times it occurs some time after food; in not a few cases the pain occurs several hours after food has been taken; the movements of the stomach, the increasing acidity of the gastric juice, and the passage of the stomach contents through the pylorus are probably the cause of it. In some cases, for example when the ulcer is associated with continuous hypersecretion (Reichmann's disease), and also, it is said, in cases of syphilitic ulcer, the pain also occurs at night or early in the morning when the stomach should be empty; the hyperacid gastric juice acting directly on the ulcerated surface is most likely the cause of this, for a small quantity of food, the protein of which binds the free hydrochloric acid, often relieves this nocturnal pain. Palpation and slight pressure of the epigastrium increase the pain, and elicit it at the time when it does not occur spontaneously, a feature which is of diagnostic importance in the differentiation of gastric ulcer from gastric neuroses.

The duration of the pain varies; often it lasts for several hours, in fact till the contents of the stomach have passed the pylorus, or have been vomited, when it more or less quickly disappears. The pain is also influenced by various circumstances, as for instance by the posture of the patient; it is less when the patient lies down, and it is aggravated by sudden movement or by undue muscular exercise or fatigue; some patients find relief when they lie on the abdomen, on the back, or on one or other side. Some observers are inclined to attribute this to the position of the ulcer, that position of the body being least painful which prevents the gastric contents from coming into contact with the ulcer. Gerhardt, however, regarded these effects of posture on the gastric pain as signifying the existence of perigastric adhesions.

The spinal pain has the same character as the epigastric, and the patient often states that it passes right through from the epigastrium to the spine. With the spinal pain, which, as first pointed out by Ross and later by Dr. Head, is a referred or somatic pain, there is often pain along the intercostal nerves, occasionally radiating to the shoulder, arm, and forearm. Ross regarded the localised epigastric pain as originating in the stomach itself—splanchnic pain—and as corresponding exactly with the site of the ulcer; this, however, is apparently not correct.

The pain may be absent for days and even weeks, and then recur; with the cicatrisation of the ulcer it gradually disappears, but, though in these cases there is no spontaneous pain, it can be elicited, as a rule, by pressure on the epigastrium.

As gastric ulcer is often accompanied by dyspeptic symptoms; besides the local pain, there is sometimes a dull pain, which is more diffuse and persists for days. In some few cases pain is absent. Associated with the epigastric and dorsal pain, there is tenderness on pressure, and as pointed out by Dr. Head and by Dr. J. Mackenzie, hyperaesthesia of the skin in corresponding situations. The hyperaesthetic area in the epigastrium is generally sharply defined, about half to one inch in diameter, and situated most frequently between the ensiform process and the left costal margin (84 per cent), occasionally further to the left (9 per cent), to the right (4 per cent), or below the umbilicus (3 per cent). The dorsal area of hyperaesthesia is somewhat less frequent than the epigastric area; it is a sharply defined area, varying in position from the 7th to the 12th dorsal spine, most commonly about the 11th, and more often to the left of the middle line than the right. Associated with the epigastric hyperaesthesia there is, as Dr. Mackenzie has shewn, a similarly localised muscular hyperalgesia and a marked exaltation of the musculo-cutaneous reflex.

The site of the pain and epigastric hyperaesthesia in their relation to the position of the stomach and to the localisation of the ulcer are very interesting. In 8 cases of low-seated hyperaesthesia Wirsing noted five instances of decided gastroptosis, while in 20 other cases of gastroptosis a change in the position of the pain was not noted, so that in downward displacement of the lesser curvature the pain of the ulcer was localised, not where the upper border of the stomach was, but where it ought to have been. Similarly Dr. Mackenzie has pointed out that the position of the hyperaesthetic area remains constant even with the most extensive respiratory movements of the stomach. The localisation of the epigastric pain would also appear to have some relation to the situation of the ulcer. In a number of cases in which it was possible to institute a comparison either by operation or by autopsy, Dr. Mackenzie found that pain localised in the upper part of the epigastrium corresponded with ulcers at the cardiac end of the stomach, low in the epigastrium with pyloric ulcers. As healing of the ulcer occurs, and the pain gradually lessens in intensity, there is a simultaneous diminution in the extent of the hyperaesthetic area, which finally disappears. Most observers are agreed on the value

of this hyperaesthesia as a manifestation of gastric ulcer; but recently Dr. B. Dawson has thrown doubt on its significance by pointing out that it is not invariably present in ulcer, and that it may occur in some other disorders of the stomach, as in anaemic and neurasthenic dyspepsia, although when present in such conditions it disappears more rapidly, and in so doing does not exhibit the contraction noted in cases of healing ulcers.

The precise mechanism of the pain in gastric ulcer has received various explanations. Formerly, on the supposition that the afferent nerves of the stomach conveyed painful impressions, it was generally believed that the pain originated by the direct irritation of exposed sensory nerves in the base of the ulcer by the acid gastric contents. When the fallacy of this supposition was exposed other explanations were advanced. According to Lennander, Mansell-Moullin, and others the pain is the immediate result of the irritation of sensory nerves in the parietal subserous tissues, either mechanically by pressure or dragging, or from a secondary lymphangitis in this situation. Dr. Mackenzie, on the other hand, regards the pain, epigastric and dorsal, the cutaneous hyperaesthesia, the muscular hyperalgesia, as expressions of a viscero-sensory reflex. Abnormally powerful stimuli passing along the afferent nerves of the stomach to the spinal cord, initiated by irritation of their exposed terminal branches in the base of the ulcer, affect adjacent centres in the cord, and so stimulating sensory, motor, and other nerves, arising in this part of the cord, give origin to pain, which is referred to the peripheral distribution of the nerve, the spinal centre of which is so stimulated. As in some cases of gastric ulcer pain may be almost the only symptom, and may occur without any vomiting of food or haematemesis, a careful inquiry as to the exact situation, limitation, and the other characters described above, is of great importance in diagnosis. For illustrative cases I refer the reader to my paper on the Clinical Diagnosis of Gastric Ulcer (39).

Vomiting.—Vomiting of food alone, without haematemesis, is common in gastric ulcer; it may occur at various stages of the disease, and be caused in various ways. It may come on early in the course of the ulcer, and occur immediately after taking food, in which case it is due most likely to hyperaesthesia of the stomach. As a rule the vomiting occurs an hour or two after the ingestion of food, at the height of the paroxysm of pain which disappears with the vomiting, and is due to the irritation produced by the contact of the food with the ulcer. In such cases the vomiting seems to depend on the quality of the food taken, liquid food being less productive of vomiting than coarse undigested food; whilst in other cases the vomiting comes on late in the disease, and is due to dilatation of the stomach in consequence of the pyloric constriction. When the dilatation coexists with hypersecretion, vomiting, as a rule, occurs some time after food, and in many cases late at night or early in the morning. The vomit itself may present some features which assist in the diagnosis of the disease. If the vomiting occur immediately after

the ingestion of food, it consists of unaltered food; if it occur some time after food has been taken, the vomit consists of partly digested food in a state of fine subdivision, and has a very acid taste. The chemical examination of the vomit or its filtrate almost always shews the presence of hydrochloric acid, very often in more than the normal quantity. The microscopic examination of the vomit shews the muscular fibres, as a rule, well-digested and undigested starch-grains. The vomit in gastric ulcer rarely contains bile; the so-called "bilious vomiting" is rare in gastric ulcer.

The vomiting that occurs in consequence of dilatation of the stomach is, as a rule, characteristic enough: large quantities are voided, pale in appearance, not unlike barm, frothy, and microscopically shewing torulae and sarcinae; the filtrate almost always contains free hydrochloric acid.

Vomiting, although an important symptom of ulcer, is by no means constant. Again, the vomiting of very acid matter, containing more than a normal amount of hydrochloric acid, occurs in a good many other affections, as in simple hyperchlorhydria, anaemic acid dyspepsia, hypersecretion (Reichmann's disease), in the early stage of pulmonary tuberculosis, in the gastric crises of tabes. From a diagnostic standpoint I regard the paroxysms of pain and the localised tenderness and hyperaesthesia as more significant of ulcer than the vomiting, and even than the vomiting of blood.

Haematemesis is one of the cardinal symptoms, and occurs in about one-third of the cases (28 per cent, Brinton; 29 per cent, Witte-Müller; 47 per cent, Gerhardt; 82 per cent, Lebert). It may occasionally, especially in so-called acute ulcers (75 per cent), be one of the first symptoms noticed, and occur in persons who were either in good health without any symptoms of indigestion, or who were troubled only with so-called acidity of the stomach; more often it occurs during the course of the disease, and is preceded by pain and vomiting.

The quantity of blood vomited varies considerably; slight haemorrhages may often occur, and small quantities of blood may be vomited or passed by the bowels without the patient's knowledge. Such occult haemorrhages, as they have been termed by Boas, are of considerable diagnostic and prognostic significance, and, as that observer has repeatedly emphasised, careful examination of apparently blood-free vomit and stools should be made before the diagnosis of ulcer is set aside. For this purpose several tests are available, the guaiacum, the aloin, and the benzidin test; of these the latter is the most delicate. It is performed as follows: A piece of faecal matter about the size of a hazel-nut is mixed with water in a test-tube to form a thick emulsion; to this one-third of its volume of glacial acetic acid is added, and the mixture shaken. Then about 5 c.cm. of ether are added, and the two fluids are mixed together—not roughly shaken; on standing, the ethereal extract separates, and of this 2 c.cm. are mixed with 2 c.cm. of a saturated solution of benzidin (paradiamidodiphenyl) in rectified spirit, and 2 c.cm. of a solution of hydrogen peroxide. If blood be present in considerable quantity the

solution immediately assumes a deep blue colour; in lesser quantities the colour is less intense and green.

In other cases the blood may not be vomited, but is passed out per rectum as melaena: in most cases, however, when the haemorrhage from the ulcer is profuse, both haematemesis and melaena appear, the latter following some hours after the haemorrhage. If melaena should occur before haematemesis, most authors look upon the case as one of duodenal rather than of gastric ulcer; but in a case of acute gastric ulcer which I saw—one verified by post-mortem examination—melaena had preceded haematemesis by more than six hours. If the quantities vomited be small, the blood, intimately mixed with the food, may escape detection; if it be slowly poured out from the ulcer, the blood may remain for some time in the stomach, and, being acted upon by the gastric juice, may have the "coffee-ground" appearance, the dark-brown colour being due to the formation of haematin from haemoglobin. If the haemorrhage from the ulcer be profuse, as for instance when a small artery is eroded, then blood is vomited in large masses: it may be quite liquid and unaltered, but occasionally it contains large clots, and, though arterial, is dark in colour from the action of the gastric juice. In profuse haematemesis the patient usually has, as premonitory symptoms, a trickling sensation, or a feeling as though something had given way, a peculiar sensation of distension, a feeling of heaviness in the stomach, followed by nausea and faintness, and a peculiar taste in the mouth, occasionally a sudden stabbing pain in the epigastrium, and then without any violent effort of retching large quantities of blood are vomited: the first haemorrhage is often followed by a second and third at various intervals. The general condition of the patient is that common to all profuse arterial haemorrhages: the skin is pallid; the patient feels cold, though often the temperature, soon after the haemorrhage, may be a few degrees raised; the pulse becomes quick, small, and compressible; the patient feels faint; his voice becomes feeble; and syncope may follow an attempt to rise from the recumbent position. These symptoms continue for some days, and anaemic murmurs are heard over the heart and the large vessels.

Haemorrhage from the stomach, even if profuse, is not often fatal, unless a large vessel be eroded, when death may occur even before there is time for the poured-out blood to be vomited. In such cases the patient suddenly turns pale, faints, falls over, and perhaps with a few spasmodic movements dies within a few minutes with all the symptoms of internal haemorrhage, and after death the stomach is found to be distended with blood. In some cases of profuse haemorrhage several attacks of haematemesis occur, and the patient dies from exhaustion; in most cases, however, the patient gradually recovers from the anaemia, and the symptoms of gastric ulcer, such as the epigastric pain which may disappear after the haematemesis, return. As a rule, several haemorrhages follow the first at intervals of a few days, and then the haemorrhage may cease altogether or reappear at a later period.

Melaena.—We have already spoken of this symptom following profuse

haematemesis; if the ulcer be situated close to the pylorus it may occur alone without haematemesis. The blood in melaena is black in colour and of tarry consistency owing to the change of haemoglobin into haematin, and the formation of sulphide of iron by the action of the sulphuretted hydrogen in the lower bowel. This symptom has often to be inquired for, or can only be inferred, as the patient is not always conscious of having discharged blood: he may complain, however, of tenesmus and of having had several liquid stools; or again, that during and after the evacuation of the stool he felt faint or actually fainted; and in his appearance there may be all the signs of a profuse internal haemorrhage. In three of my own cases general convulsions occurred in consequence of melaena from gastric ulcer.

Apart from these principal symptoms in gastric ulcer there are others less characteristic and less frequent. Considering these symptoms according to the various systems we have:—

Gastro-intestinal Symptoms.—The tongue varies very much in appearance; often it is red, clean, and moist: when there is marked anaemia it is pale and flabby; in chronic cases, on the other hand, it is often found covered with white fur, which is due to the accompanying gastric catarrh. The appetite is generally good, but the patient is afraid to eat because of the pain after food: sometimes the patient has an increased appetite; in very chronic cases the appetite is often diminished. Thirst is increased, and in some cases there is an increased flow of saliva. Dyspeptic symptoms, such as flatulence, sense of fulness and of discomfort after food, acidity and pyrosis, are common, especially in chronic ulcers; constipation is usual, but in a few cases the bowels are regular; diarrhoea is very rare.

Cardio-vascular Symptoms.—In many cases nothing abnormal in the heart or blood-vessels is noticed; occasionally there is irregularity of the heart's action, and, as anaemia so often complicates gastric ulcer, we have all the vascular symptoms noticed in anaemia.

The blood invariably exhibits pronounced changes, usually of a chlorotic character. The red blood-corpuscles are diminished in number, occasionally even to two millions per c.mm. The average in 44 cases is given by Futcher as 4,071,000 per c.mm., in 51 cases by Cabot as 3,372,000 per c.mm. Greenough and Joslin, on the other hand, out of 43 cases found only 24 in which the red corpuscles were below 4,000,000. The haemoglobin in Futcher's series averaged 58 per cent, the average haemoglobin-index being 0·72. In 42 of Cabot's 51 cases the haemoglobin was below 50 per cent. In 43 of Greenough and Joslin's cases the average haemoglobin-index was 0·67. The leucocytes in Futcher's series averaged 7500 per c.mm., differential counts by Cabot shewed a slight relative increase in the small mononuclears at the expense of the polymorphonuclear neutrophils. There is a well-marked digestion-leucocytosis.

Genito-urinary Symptoms.—The urine is in many cases normal, at other times it may contain albumoses: on the other hand, owing to the in-

creased secretion of hydrochloric acid, it is often but slightly acid and may even become alkaline; this is especially the case when the stomach is dilated; the chlorides are often diminished and the phosphates increased; if the dilatation of the stomach be considerable, and the decomposition due to micro-organisms increased, the urine may contain an excess of the ethereal sulphates and of indican. The menstrual functions are, as a rule, not interfered with unless chlorosis be present or after profuse haematemesis. The occurrence of sugar is rare; albumin, on the other hand, is more frequently to be found.

Acetonuria and Diaceturia.—It is now well established that acetone and aceto-acetic or diacetic acid are occasionally found in the urine in acute and chronic affections of the digestive tract, such as acute and chronic gastric catarrh, enteritis, dilatation of the stomach, gastric ulcer. Both bodies are often found together in the urine, as acetone is formed from the former; in some cases, however, acetone alone is found, and may be detected by the smell and the various reactions (78), whilst the presence of aceto-acetic acid is easily detected by Gerhardt's reaction. In many cases the presence of these bodies gives rise to no marked symptoms; in others, however, we may find a group of gastro-intestinal and nervous symptoms, which may assume a serious character resembling the toxic symptoms in diabetic coma, and due in all probability to an acid intoxication (40). In the milder form we notice severe epigastric pain, persistent vomiting, headache, and giddiness. In the severe forms these symptoms may be associated with dyspnoea, drowsiness, coma, and epileptic convulsions. The symptoms usually set in suddenly and subside after a few days with the disappearance of the acetone from the urine. In most cases the affection is easily and early recognised by the peculiar acetone smell of the breath and of the urine. Lorenz noticed this affection in five cases of gastric ulcer, and I have observed one case: the patient, a girl aged twenty-two, who had had all the symptoms of gastric ulcer, was suddenly seized with violent abdominal pain, vomiting, and great prostration; the vomiting persisted and could not be checked by any medicine, all food and even small quantities of water were rejected; the patient had repeated attacks of syncope when she attempted to raise herself, the pulse was slow, and the temperature normal. The breath had a marked acetone smell; the urine, which was scanty, had a sp. gr. 1028, had a strong smell of acetone, and gave the aceto-acetic acid reaction with perchloride of iron; the symptoms persisted for five days, during which time the patient was fed by nutritive enemas; citrate of potash in solution was given by the mouth and injected into the rectum. The symptoms then gradually subsided and the patient made a rapid recovery.

Drs. Rolleston and Tebbs have made a series of observations on the occurrence of diacetic acid in the urine in cases of gastric ulcer. They found diaceturia in 33 out of 38 cases of gastric ulcer in females in whom no food was given by the mouth. Gerhardt's reaction in these cases appeared on an average on the second or third day of absolute starvation

or rectal feeding, and it was apt to disappear as soon as feeding by the mouth was resumed. In four cases of gastric ulcer in males the reaction either did not occur at all or only appeared after prolonged starvation. It would appear that the exciting cause which determines the appearance of diaceturia is starvation or defective absorption, and it makes no difference whether the patient be absolutely starved or fed per rectum. The addition of glucose to the nutrient enemas did not diminish the diaceturia; in fact, in one of the male cases this was responsible for its appearance. Glucose given by the mouth, however, was rapidly followed by the disappearance of the acid from the urine. In cases with severe and persistent vomiting there is usually a well-marked reaction. Examination of the blood of certain of these cases by Dr. Golla failed to reveal any appreciable diminution in the CO_2 content.

The *temperature* is normal or even subnormal if the nutrition suffer much. Rise of temperature denotes some complication or some intercurrent disease. Slight fever lasting for a few days to a week not uncommonly follows the vomiting of blood.

Nervous symptoms are common enough in gastric ulcer. Headache, vertigo, insomnia, and attacks of syncope are not uncommon; nervous symptoms are often present also when neurasthenia, melancholia, or hysteria complicates the affection. The superficial reflexes vary; in neurotic or hysterical subjects they are often increased, and in such cases the abdominal and epigastric reflexes may be exaggerated. Tetany is an occasional and grave complication of gastric ulcer; it appears to be somewhat more frequent in males, and is almost invariably associated with dilatation of the stomach due to the presence of a pyloric ulcer or a cicatricial pyloric stenosis. The attacks have followed the washing out of the stomach, and in some cases it is sufficient to pass a soft stomach-tube or merely to rub the skin of the epigastrium to induce an attack. It is generally ascribed to an auto-intoxication by toxic products of decomposition in the dilated stomach. In such cases gastro-enterostomy may be called for to relieve the dilatation.

The general nutrition in many cases remains good for some time; but with the duration of the disease it becomes much impaired, there is loss of flesh and the muscles feel flabby, the patient is pale, has an anxious appearance, the eyeballs are sunken, and the whole expression denotes much suffering. In younger persons the aspect is most commonly that of anaemia simply; in older persons it is cachectic rather.

The physical examination in many cases reveals nothing but the tenderness of the epigastrium and an accompanying cutaneous hyperaesthesia of the region; in very chronic cases, when the patient is much emaciated, a fibroid cicatrix may sometimes be felt as a band or as a slight intumescence, and in those rare cases in which a localised abscess has formed between the stomach and the adjacent parts (abdominal wall, spleen, liver, etc.), a distinct tumour, painful on percussion and pressure, may perhaps be felt. Gerhardt distinguishes four varieties of intumescence which may be met with in old ulcers:—(1) The cicatricial

base of the ulcer, to be recognised by its plate-like feel, its sensitiveness, and its frequency; (2) the pylorus, hypertrophied from spasmodic contraction; (3) an encapsuled abscess; and (4) portions of organs adherent to an old ulcer which may form plugs protruding into the stomach and become inflamed and indurated; in this way massive painful tumours are formed which may even increase slowly in size. In such cases chemical examination of the gastric contents may be of considerable value. In 16 cases of gastric ulcer with a palpable tumour in which the precise conditions were ascertained after death Reinhard found that the tumour was due to hypertrophy of the pylorus in 6, to adhesions in 6, to perigastric abscess in 1, and to the presence of foreign bodies in the stomach in 3, hair-ball, chalk, and vegetable fibres. The discrimination of such an intumescence from the tumour of cancer is, as a rule, not difficult when the history and symptoms are carefully considered. Marked dilatation of the stomach can be ascertained by physical examination.

Alterations of the Functions of the Stomach in Gastric Ulcer.—*Hyperacidity.*—For some time great stress was laid on the increased amount of hydrochloric acid in cases of gastric ulcer, and no doubt free hydrochloric acid is found in the majority of cases. In many cases the increased acidity is found soon after food is taken, and, at the height of the digestion, 0·3-0·35 per cent of hydrochloric acid may be found in the contents of the stomach. In some cases the fasting stomach is found to contain a small quantity of fluid which contains hydrochloric acid (hypersecretion of Reichmann); in these cases there is a continuous secretion of acid gastric juice, and these are probably the cases in which patients complain of pain in the epigastric region late at night or early in the morning when the stomach should be empty. In about half the cases of ulcer the quantity of hydrochloric acid is normal, in a few cases the acid is diminished (sub-acidity); this occurs in chronic cases when the ulcer is complicated by anaemia or chronic gastric catarrh; and in some cases in which the stomach is dilated; though an excess of hydrochloric acid is perhaps more often noticed in dilatation following ulcer.

In many cases the hyperacidity can be made out by examination of the vomited matter; to use the stomach-tube in order to examine the gastric juice and the contents of the stomach after a test-meal is only permissible when there has been no haematemesis; the introduction of the stomach-tube may produce not only haematemesis but, if the ulcer be acute and its floor thin, perforation also. At the same time the passage of a soft tube with care cannot be nearly so dangerous as the strain of vomiting. In making any determinations of the hydrochloric acid content of the gastric juice, test-meals removed by means of the stomach-tube after a definite interval must be employed; and it must be remembered, especially in view of the variable results obtained, that the Ewald's breakfast is a relatively poor stimulus, and that the response to the stimulus of feeding varies at different times. In brief, any statements with regard to the acid values in gastric ulcer and other disorders must be based on several observations made under identical conditions in

each case. The chemical tests for free hydrochloric acid are described in the article on "General Pathology of Digestion" (p. 279).

The secretion of *pepsin* does not undergo much alteration in gastric ulcer, occasionally it is increased; in old ulcers with marked emaciation or dilatation of the stomach, it is often diminished.

The *motor* functions of the stomach are not interfered with by the ulcer unless there be marked dilatation, or some such complication as pulmonary tuberculosis or chlorosis; though in a few cases the movements of the stomach are diminished (Korczynski and Jaworski).

Absorption from the Stomach.—Zweifel gave iodide of potassium in gelatin capsules, and determined the length of time that elapsed before he could detect iodine in the saliva and in the urine; the absorption was generally prolonged but very slightly, except in the early stages of ulcer, when it may be more prolonged.

When the stomach is dilated we often find bacterial decomposition, sometimes with the formation of nitrogen, oxygen, carbonic acid, occasionally of hydrogen, sulphuretted hydrogen, and marsh gas.

Complications and Sequels of Gastric Ulcer. — *Dilatation of the Stomach.*—The usual cause of dilatation of the stomach in gastric ulcer is the narrowing of the pylorus due to the cicatrisation of an ulcer situated at or near the pylorus. It may also be produced by spasmodic stenosis of the pylorus, an occasional complication of pyloric ulcers. In these latter cases the dilatation is generally associated with hypersecretion, and the manifestations are severe cramp-like pain in the epigastrium at the height of digestion, increased and visible peristalsis, and the vomiting of large quantities of very acid fluid contents. During the spasm the pylorus may be felt to harden and feel like a tumour, and eventually there may result a permanent intumescence in the pyloric region from hypertrophic thickening of the pyloric musculature.

Occasionally simple or atonic dilatation of the stomach, without stenosis of the pylorus, due to accompanying gastric catarrh or to muscular weakness may be present. As dilatation of the stomach is described in a separate article (p. 522) no further reference to its symptoms or treatment need be made here.

Hour-glass Deformity of the Stomach.—The manifestations to which it gives rise are most commonly those of dilatation of the stomach, but occasionally the following characteristic signs of the condition are noted:—(1) In washing out the stomach part of the fluid is lost, and fails to return through the tube (Wölfer); (2) the wash water returning from the stomach quite clear will suddenly become turbid from the reappearance of stomach contents (Wölfer); (3) after the stomach has been apparently emptied by the stomach-tube, a splashing sound may be elicited on palpation of the pyloric portion (paradoxical dilatation of Jaworski); (4) on inflating the stomach with air or CO_2, the peculiar form of the organ may be visible or palpable; (5) during the process of inflation the cardiac portion may become first distended, and then gradually subside with concomitant distension of the pyloric portion

(Eiselberg); (6) during the above-mentioned change gushing, bubbling, or sizzling sounds may be heard at a point distinct from the pylorus.

Perigastric Adhesions. — Clinically the existence of such adhesions may be rendered manifest by the production of various deformities, as dilatation from kinking of the pyloro-duodenal junction, hour-glass deformity in which the adhesions are situated anteriorly, general contraction of the stomach (the Fenwicks), and approximation of the two orifices; and by various functional disturbances of all degrees of severity. There is pain after food, of a dragging kind, about the pyloric region or less commonly over the fundus of the stomach; it is always worse after a bulky than after a small meal; it is increased by the erect posture, by exercise and particular movements, such as raising the arms, particularly the left; and it is relieved by rest in the recumbent posture, and often also by wearing an abdominal belt. In consequence inability to follow the avocations of life is complained of. Where there is dilatation periodic vomiting of large quantities of fluid or semi-fluid contents occurs; the vomit as a rule contains free hydrochloric acid, often in excess. There is also some loss of flesh. Tenderness on pressure over the epigastrium is generally present.

Perforation of the stomach is common, and is the most grave complication of gastric ulcer. It occurs much more frequently in acute ulcers or in an early stage of the chronic ulcer; it often causes the death of the patient by peritonitis; in rare cases a small perforation may heal, and the patient may completely recover; in other cases it may lead to the formation of an abscess, and in other cases, again, to a fistulous communication between the stomach and other organs.

Perforation into the General Peritoneal Cavity.—This, according to Welch, happens in about 6·5 per cent of all cases of gastric ulcer, an estimate which is probably as nearly correct as can be obtained. It occurs more commonly in young females; but allowing for the greater frequency of ulcer in females, perforation is relatively more common in males, especially over forty years of age. As already indicated, this untoward complication is found chiefly in ulcers situated on the anterior wall of the stomach. Lindner and Pariser estimate that of 200 ulcers, 190 will be located on the posterior wall, of which 4 will perforate, 10 will be on the anterior wall, and of these 8½ will perforate. According to Brinton 70 per cent of all perforations are on the anterior wall, 21 per cent on the lesser curvature, and 9 per cent on the posterior wall. The Fenwicks, from a series of operations and post-mortem cases, conclude that whereas the acute ulcers usually perforate the comparatively thin coats of the cardiac portion of the viscus on the anterior wall near the lesser curvature, the chronic ulcers are most prone to perforate in the pyloric region on the posterior surface near the upper margin. Perforation may be multiple; this is found in 20 per cent of the recorded cases. Perforation may occur in cases in which there have been no definite symptoms of ulcer, or in which almost all symptoms have been absent and the patient apparently in good health. In other cases, however, perforation takes

place after local pain, vomiting, and even haematemesis have existed for some time.

The symptoms of perforation are usually characteristic enough, unless death from collapse take place within a few hours after its occurrence. The first symptom is sudden pain of severe and agonising character, which may be situated at first in the epigastrium, and then become diffused over the whole abdomen; or it may be seated rather over the right iliac fossa, and simulate the perforation of appendicitis or pyosalpinx; this I have seen in not a few cases. The pain may be continuous, or it may have the character of a severe colic. Along with the pain come the symptoms of collapse; the pulse becomes small, quick, and thready, but occasionally for some little time following perforation it may be slow and of moderate tension; the face is pale, and has a pinched, drawn appearance (*facies hippocratica*): the surface of the body is covered with cold, clammy perspiration; the voice becomes feeble, and the respirations frequent and superficial, and after a short time costal in character. The temperature, at first subnormal, becomes raised if the patient live for some time and peritonitis supervene. The tongue becomes dry; thirst is increased; vomiting is an inconstant symptom occurring in 40 per cent (Finney), or 29 per cent (the Fenwicks), though often there is much retching; constipation is marked; the urinary excretion is diminished, and with the onset of peritonitis micturition becomes painful, or may be entirely suppressed; the urine may contain albumin and casts. On physical examination, besides the general features above noticed, we find the abdomen, as a rule, distended; but in a few cases, as pointed out by Wagner and myself, it may be retracted by the spasm of the abdominal walls; it is very sensitive to the touch, with local pain over the epigastrium or right iliac fossa. Percussion gives a tympanitic note all over the abdomen, with absence or considerable diminution of the liver dulness from the accumulation of gas between the liver and diaphragm; this has been described as a pathognomonic sign of perforation, but it may be absent if adhesions exist between the stomach and liver; on the other hand, we may have this sign without perforation in simple tympanites if coils of intestine distended with gas find their way between the diaphragm and the liver, and it may occur in simple non-perforative peritonitis. If much fluid pass out from the stomach the percussion note over the lateral and lower parts of the abdomen may become dull (a very rare occurrence); this symptom may also be noticed subsequently when peritonitis is well established. If the patient do not die from collapse, which happens in about 4 per cent, during the first six or twelve hours, symptoms of peritonitis set in; the temperature may rise to 102° or 103°; the pulse becomes harder; the breathing becomes very shallow; retching and hiccup occur, and death follows from exhaustion or collapse. In some cases the pain becomes less severe, and the patient may seem better, yet death occurs in a short time.

Most cases of perforation from gastric ulcer would end fatally unless

dealt with surgically: in a few cases, however, if the perforation be very small, and but little of the contents of the stomach escape into the peritoneal cavity, the opening may be closed by fibrin, and the patient may eventually recover. Perforation of an ulcer of the stomach into the general peritoneal cavity may occasionally be simulated by perforative appendicitis, and it is important to remember that both accidents have been met with simultaneously. Another condition which may prove a source of error is acute haemorrhagic pancreatitis. In women, as pointed out by Mr. Waring, the symptoms of a perforated ulcer may be closely simulated at or about the time of menstruation; the symptoms commence suddenly with a sharp pain in the abdomen, which may be distended, tender, and immobile, the pulse is rapid, and the temperature often subnormal. The pain may be chiefly referred to the epigastrium simulating gastric or duodenal perforation, or to the right iliac fossa simulating appendicular perforation. In most cases the general appearance of the patient is not so desperately bad as in perforative peritonitis, and almost all have recovered, although I have seen one fatal case in which haemorrhage into one of the ovaries was the sole feature of the autopsy.

Localised Abscess the Result of Perforation.—In cases in which but a small quantity of the stomach contents has escaped, and in which before perforation adhesions, more or less firm, have been formed between the stomach and the neighbouring organs, we may have a local peritonitis with subsequent formation of an abscess. Whilst the symptoms at first are those of perforative peritonitis the further course varies according to the situation of the abscess; in many cases the general and local symptoms of peritonitis diminish; the abdomen is less distended and less painful, and the appetite improves; but strength is not regained, the pulse remains quick, the fever assumes a remittent type, rigors are occasionally observed and there may be profuse nocturnal sweating, and the patient eventually dies from exhaustion, or by way of pyaemia or of pylephlebitis. In other cases the patient improves considerably for a time till the abscess bursts into the peritoneal cavity, when he succumbs to general peritonitis; this is the case when the abscess forms behind the stomach, or between the stomach and the spleen, or is encysted in the mesentery; in rarer cases still the abscess after having perforated the diaphragm may open into one or other of the thoracic organs (pericardium, pleura, lung, mediastinum). In other cases, however, the pus from the abscess may, either directly or by a fistulous tract, be discharged outside or become accessible to operative measures, and the patient recover (*vide* article on "Subphrenic Abscess").

Perforation of a gastric ulcer, without the intervention of an abscess, may take place either into a hollow or solid organ to which the stomach has previously become united by adhesions. If perforation take place into a part of the intestine, a *bimucous fistula* results (39); the communication may be made between the stomach and the duodenum, or any other portion of the intestine; most commonly it is made directly with

the transverse colon (gastro-colic fistula), and the condition may be diagnosed by the rapid passage of food through the digestive tract.

If adhesion take place between the stomach and diaphragm the ulcer may perforate into the left pleural cavity or, if the two layers of the pleura are adherent, into the base of the left lung; and thus *pyopneumothorax* or *gangrene of the lung* may be established. According to the Fenwicks eleven examples of this complication are on record: in three the perforation was followed by empyema, in four by pyopneumothorax, and in four by gangrene of the lung. Some five cases are recorded in which the pericardium has been perforated, with the result of pneumopericardium and gangrenous pericarditis. Debove and Renault give a brief description of some of the recorded cases. A sudden epigastric pain is followed by dyspnoea and cyanosis; the apex beat of the heart cannot be felt, the cardiac dulness is replaced by a tympanitic note, pericardial friction may be heard, the internal heart sounds may be accompanied by a loud metallic timbre, the pulse becomes rapid and feeble, and the patient dies within a short period after the occurrence of the perforation. In yet rarer cases the pericardium may become adherent to the heart, and the ulcer of the stomach, by an extension of the ulcerative process, may burst into the heart. According to Debove and Renault only four such cases are on record; the initial symptoms consisted of faintness and oppression at the chest, where the subsequent haemorrhage was comparatively slight at first, haematemesis and melaena were prominent symptoms, as in Brenner's case in which they lasted for three days. On the other hand, profuse haemorrhage proving rapidly fatal was not associated with vomiting, as in Finny's case.

General Subcutaneous Emphysema.—Emphysema of the subcutaneous and subserous tissues is a rare complication. About 9 cases have been observed; in some it appears to have followed perforation into the posterior mediastinum, in others the gas would seem first to have distended the peritoneal cavity and subsequently to have insinuated itself into the subserous tissues at the edge of the ulcer or at some spot where the peritoneum had been damaged.

Another complication of gastric ulcer is the supervention of *cancer*. This coincidence was already known to Dietrich and Brinton, who drew especial attention to the subject; according to Lebert 9 per cent of all cases of cancer of the stomach owe their origin to simple ulcer of the stomach, but according to Rosenheim only 6 per cent; Haeberlin, again, reduces the number to 2-3 per cent. The observations of Hauser have thrown some light on the implantation of cancer on the ulcer; for he noticed masses of epithelial cells amidst the fibrous tissue at the borders of the ulcer, from which cells cancer may take its origin. The naked eye and histological appearances of the cancer thus formed do not differ from those of other cancers in the stomach; but the clinical features of such cancers, as a rule, are for a long time those of gastric ulcer, till eventually a tumour appears, and with it cancerous cachexia; yet even then the gastric juice still shews the presence of hydrochloric acid, and sometimes

even of hyperacidity. In other cases the history of a case of cancer with the ordinary symptoms indicates that an ulcer had existed years before (39). (*Vide* "Tumours of Stomach," p. 495.)

Course and Duration of Gastric Ulcer.—From the symptoms of gastric ulcer given above, it is evident that the disease shews an extreme diversity both in its features and in its subsequent course and terminations. Anatomically we distinguish between acute and chronic ulcers, and clinically the same classification has been adopted very rigidly by the Fenwicks; but, as gastric ulcer may exist a long time without producing any symptoms, we must not assume that, if haemorrhage from the stomach or sudden perforative peritonitis occur in a person presumably healthy, we have to do with an acute ulcer; it is more likely that the ulcer has existed for some time in a latent state. Many cases of gastric ulcer (according to Cruveilhier about 80 per cent) end in recovery; in many there are remissions which may occur at intervals of months or years; and cases are not infrequent in which ten and twenty years after the occurrence of the first symptoms the characteristic signs of gastric ulcer are noticed again; no doubt in these cases the old ulcer had cicatrised, and either had broken out afresh or given place to a new ulcer: in other cases, some time after the disappearance of the characteristic symptoms the signs of dilatation of the stomach appear; in others again, as stated above, either cancer or one of the other complications supervenes. Clinically the following forms may be distinguished:—(i.) *Acute Haemorrhagic Form.*—Either without previous symptoms or after symptoms of very short duration profuse haematemesis occurs. The case may end fatally, symptoms of chronic ulcer may supervene; or, in a few cases, no further symptoms may appear. (ii.) *Acute Perforating Ulcer.*—Either without any previous symptoms or after slight gastric disturbance, sudden perforation into the abdominal cavity occurs, in a very large majority of cases with a fatal termination. (iii.) *Chronic Dyspeptic Form.*—In this there are marked dyspeptic symptoms, with moderate pain and occasional vomiting. (iv.) *Chronic Gastralgic Form.*—Gastric pains predominate; vomiting may or may not occur. (v.) *Chronic Haemorrhagic Form.*—This resembles the chronic dyspeptic form, with haematemesis superadded.

Some authors speak also of (vi.) a *cachectic form*, in protracted cases, in which emaciation, anaemia, and a cachectic appearance—not unlike that seen in cancer—appear; of (vii.) a *stenotic* form, with symptoms of dilatation of the stomach; and of (viii.) a *recurrent* form in which, long after apparent healing of the ulcer, the symptoms recur.

Diagnosis.—In many cases of gastric ulcer the diagnosis is easy; in others the diagnosis can only be arrived at after a careful analysis of all the symptoms, and after watching the patient for some time; whilst in not a few cases a definite diagnosis is impossible, as most of the symptoms are absent. None of the symptoms mentioned above is absolutely characteristic of gastric ulcer, and all may occur in other affections of the stomach and in other diseases.

Of the several cardinal manifestations, pain, vomiting, haemorrhage,

and hyperacidity, the pain with its peculiar features and concomitant cutaneous hyperaesthesia and muscular hyperalgesia is certainly the most characteristic. Dr. Dawson maintains that such may be present in anaemic dyspepsia and in neurasthenic dyspepsia; this, however, is not my experience.

Haematemesis, one of the most striking of these cardinal manifestations, and one on which a diagnosis is very generally based, is apt to be misleading if its significance is not duly considered in the light of other manifestations. It may follow the swallowing of blood coming from the nasopharynx, the gums, the tonsils, or in infants from a cracked nipple; in such fictitious haematemesis, which may be accidental or intentional, the quantity of blood vomited is generally small and the act may be frequently repeated. Bleeding from the mucous membrane of the stomach itself may occur in a number of conditions other than from gastric ulcer, when its true significance becomes apparent only by a careful consideration of other manifestations of the morbid state in question. Thus, it may, in rare instances, follow the seizures of general paralysis depending on haemorrhages into the gastric mucosa, analogous to those which have been produced experimentally by various lesions of the nervous system. According to Leo and Lancereaux, it may be part of a true neurosis. It may be a manifestation of the general haemorrhagic tendency in splenic anaemia, scurvy, purpura, and rarely in haemophilia (Litten). It has been noted in cases of cholelithiasis (Fleiner and Minkowski), after operations on the gall-bladder and for gall-stones (Dahler, Schmidt, Reinhard) and other abdominal operations (post-operative haematemesis (110)), a possible explanation of which is afforded by the experimental work of Dalla Vedova. In various septic processes, septic inflammations of the urinary passages (Guyon), appendicitis (Dieulafoy, Böckel, Ewald, Fuchsig), in acute pneumonia (Dieulafoy, Fraenkel) probably as a consequence of infective embolic erosions or inflammation of the lymphoid follicles in the gastric mucosa. In acute and chronic gastric catarrh bleeding occasionally occurs (Lambotte, Ewald), and it has been noted in dilatation of the stomach without apparent cause, and in such cases is by Möser ascribed to nervous hyperaemia from pyloric stenosis. In cirrhosis of the liver and in chronic heart disease haematemesis may follow the rupture of dilated and varicose veins at the lower end of the oesophagus or in the fundus. Small miliary aneurysms may be the cause of severe and even fatal haemorrhage (Hirschfeld); and direct injury to the gastric mucosa is an occasional cause, as in Heilbron's case in which haematemesis followed the swallowing of a splinter of glass contained in a glass of beer.

Haemorrhagic Erosions and *Gastrostaxis.*—A considerable number of cases of sudden profuse and even fatal haemorrhage from the gastric mucosa have been recorded in which subseqent inspection has revealed scattered superficial erosions or an apparently intact mucosa. Erosions which, as we have seen, may arise from a small capillary haemorrhage into the mucosa or from acute inflammation of the lymphoid follicles, may be

the source of very severe and rapidly fatal haemorrhage when they extend deeply and erode some considerable branch of the coronary artery (Dieulafoy, Hood, Steven, Mayo Robson). In the majority of cases, however, they apparently have but little clinical significance; although Einhorn, Pariser, and others have formulated a symptom-complex of which they represent the hypothetical and as yet unverified pathology. This disease — haemorrhagic erosion of the stomach — is said to be characterised by pain occurring from one- to three-quarters of an hour after food, of variable intensity and diffused over the stomach, without hyperaesthesia or dorsal tenderness; by occasional vomiting of material, which may contain altered blood (coffee-ground vomit); normal or subnormal acidity of the gastric contents and the presence of blood-stained fragments of mucosa in the water of washings from the empty stomach.

Dr. Hale White, who has introduced the term *gastrostaxis*, has long been of opinion that many cases of haematemesis in young anaemic females, which would commonly be attributed to gastric ulcer, do not belong to this category, but represent an independent disease of which the principal manifestation is haematemesis due to an oozing of blood from the mucous membrane. Many such cases known to the older writers were formerly regarded as examples of vicarious menstruation—a possibility reasserted by Küttner as recently as 1897. In none of Dr. Hale White's cases, however, was any monthly periodicity noticeable. In addition to haematemesis, the patients suffer from gastric pain and tenderness, and vomiting, lasting over a considerable period — perhaps years; they appear better nourished than sufferers from gastric ulcer. The condition is rarely fatal. As regards the source of the haemorrhage Dr. Hale White's observations are entirely negative, but it would appear not improbable that the condition of gastrostaxis depends on the formation of the minute deep pore-like erosions described by other observers.

Anaemic Dyspepsia.—Chlorosis may be associated with marked dyspeptic symptoms, such as loss of appetite, pain after food, and occasional vomiting. The absence of localised pain, the irregularity of the vomiting and its independence of meals, will often enable us to distinguish this condition—due to anaemia and hyperaesthesia of the stomach—from gastric ulcer; on the other hand, as gastric ulcer often occurs in chlorotic women, it will be well, if in doubt, to treat the case as one of gastric ulcer, and to notice the effect of the treatment: if the symptoms be due to gastric ulcer the appropriate treatment may give relief in a few days; if, on the other hand, they are due to chlorosis, a good effect should follow the administration of iron.

Gastric Catarrh.—Acute catarrh can scarcely be confounded with gastric ulcer. Subacute and chronic gastric catarrh have the following distinguishing features: the pain is not localised; it is not so severe as in ulcer; it is not increased by pressure; it is generally accompanied by flatulence, distension, or discomfort for hours after food; vomiting occurs

at irregular intervals; the vomit contains mucus, and there is no hyperacidity of the gastric juice; haematemesis is extremely rare, and if it occur the quantities of blood are small. The appetite is diminished, and there is frequently a desire for highly seasoned foods. The tongue is coated, flabby, and foul.

Disorders of Secretion — Hyperchlorhydria and Hypersecretion. — Both these conditions may present some difficulty in diagnosis. There is pain, heartburn, pyrosis, from one to two hours after food, with occasional vomiting which affords relief from the pain. The vomit or the gastric contents shew an increase in free hydrochloric acid. Haematemesis, however, does not occur, and it is in such cases that examination of the vomit and faeces for occult haemorrhage is especially important. The pain, though greatest in the epigastric region, is diffused over the whole stomach area, there is rarely tenderness on pressure unless pyloric spasm coexists, when the pylorus may feel sore, otherwise pressure generally relieves the pain. Dorsal points of tenderness and epigastric cutaneous hyperaesthesia are generally absent. In both hyperchlorhydria and hypersecretion the pain reaches its maximum at the height of digestion, somewhat later than in ulcer, and is relieved by food for the time being. In hypersecretion of the continuous type (Reichmann's disease) there is nocturnal pain in the empty stomach, often associated with vomiting of very acid fluid, and from the fasting stomach a small quantity of acid gastric juice can always be obtained. This condition is frequently associated with dilatation and pyloric spasm. Some writers, as the Fenwicks, in this country regard Reichmann's disease as invariably due to gastric ulcer, that it is a complication of an ulcer situated in the pyloric region; in some cases this association undoubtedly exists, but I have observed several cases in which the presence of ulcer could be eliminated.

Hyperaesthesia and gastralgia may simulate gastric ulcer in the intensity of the pain. They differ in the following points: the pain may come on immediately after food and may be more intense after liquids than solids, or it may appear irregularly without any relation to food. It is felt over the whole stomach area and is frequently relieved by pressure. Vomiting is an occasional occurrence and is followed by some amelioration of the pain. Haematemesis does not occur.

Gastric Crises.—Locomotor ataxia may be associated with severe attacks of abdominal pain and vomiting which may simulate ulcer. These crises, however, often occur independently of food; they are rarely associated with haematemesis. Both Charcot and Vulpian mention such a complication; the pain is felt over a wider area and is not relieved by vomiting. On the other hand, there are the cardinal symptoms of locomotor ataxia.

Cancer of the Stomach.—The most important points are: that cancer occurs in most cases after the fortieth year; the pain is more continuous and, although increased by the taking of food, persists when the stomach should normally be empty; the appetite soon fails; quite early there may be a distaste for particular foods, especially meat;

vomiting occurs later in the disease, and has rather the character of the vomit of dilatation of the stomach; haematemesis is not so profuse, and has the coffee-ground character; occult haemorrhage is more constant and characteristic of cancer than of ulcer. The presence of a tumour in or near the epigastric region is the most characteristic sign of cancer, yet it must be remembered that in some cases of cancer no tumour can be felt; on the other hand, in ulcer of the stomach, as already stated, a fibroid cicatrix or a local abscess may simulate a tumour. Equally important in cancer are the usual absence of hydrochloric acid in the stomach contents and the presence of lactic acid and of the Oppler-Boas bacillus, the steady loss of weight, and the cachectic appearance; we must not forget, however, that ulcer in older subjects may be attended by more or less cachexia, but in cancer the general symptoms as a rule appear early, in ulcer very late. In some cases of cancer, however, especially if situated at the cardiac end, or diffusely infiltrating the stomach, the appearance of the patient is more anaemic than cachectic; and, again, the subjects of cancer under appropriate treatment may shew a distinct increase of weight for a time. It has been shewn by a number of observers that whilst in gastric ulcer there is a well-marked digestion-leucocytosis, this is wanting in gastric cancer. According to Salomon and others (5A), the presence of more than 0·5 per mille of albumin (Esbach) and of urea in the washings of the fasting stomach indicate cancer.

Ulcer of the Duodenum.—The differential diagnosis can only be made in a few cases in which there is melaena, and a deeper and less severe pain in the right hypochondriac and umbilical regions, occurring three to five hours after food without the dorsal pain. Vomiting is inconstant; it bears no relation to the ingestion of food and affords no relief to the pain; according to Boas the vomit may be alkaline, contain bile, and digest fibrin. It occurs more frequently in men of middle age.

Gall-Stones.—In the majority of cases the distinction between biliary colic and the gastralgia of gastric ulcer is easy; but in some cases of gall-stones the main symptom at first is a severe local pain with or without vomiting, and jaundice or the definite symptoms of biliary colic are deferred. The chief points of distinction are: (α) As regards the pain; in hepatic colic the pain comes on suddenly, some hours after a meal or at night; especially after an indiscretion of diet, a muscular effort, or mental shock or excitement. The pain is very severe, and is rather colicky than burning in character as in gastric ulcer; it is not situated in the epigastric region (except when the gall-stone obstructs the common duct near its termination, in which case jaundice is invariably present); it lasts for many hours and subsides suddenly; it is associated with a pain over the right shoulder-blade, and on the subsidence of the pain, or soon after, the epigastric region often ceases to be tender to touch or pressure; at times, however, the pain on pressure persists. (β) As regards vomiting, this occurs soon after the beginning of the attack, and persists for some time; at first every kind of food is

vomited very soon after it is taken; the vomited matter shews no hyperacidity. (γ) A rise of temperature with or without a shiver is characteristic of gall-stones. (δ) Profuse sweating and great prostration are also in favour of gall-stones. (ε) The conjunctivae shew at times a distinctly icteric tint, and the urine may contain small traces of bile even though there be no apparent jaundice.

A movable or displaced kidney may produce severe gastralgic pains and vomiting, and so simulate a gastric ulcer. The situation of the pain, the absence of epigastric tenderness and hyperaesthesia, and of haematemesis, together with the condition revealed by a bimanual examination, will, as a rule, clear up the diagnosis. Usually symptoms of neurasthenia are present in these cases.

The diagnosis of complications has already been spoken of under Symptomatology; for the distinction of dilatation of the stomach due to ulcer from dilatation due to other causes see p. 522.

Diagnosis of the Seat of the Ulcer.—This in many cases is not possible, and no great reliance can be placed on the situation of the pain: it is said that an ulcer on the posterior surface gives rise to pain situated in the back rather than in the epigastrium, and is increased by the recumbent posture; here also it is said to give rise more often to haemorrhage than if situated on the anterior surface; in the latter situation it is said to give rise to pain to the right or to the left of the middle line in the epigastrium—pain which is lessened when the patient lies on the left side. Perforation into the abdominal cavity is more common with ulcer on the anterior surface; ulcer at the pylorus is said to cause pain in the middle line or to the right of the middle line, and the pain does not occur till some time after the ingestion of food; cicatrisation of the ulcer in this situation causes dilatation of the stomach. Dr. J. Mackenzie, from observation of the site of the ulcer in a few cases, finds that pain and hyperaesthesia high in the epigastrium correspond with ulcers in the cardiac portion of the stomach, whilst in pyloric ulcers pain and hyperaesthesia are referred to the lower part of the epigastric area.

Prognosis of gastric ulcer treated by dietetic and drug methods is favourable, the more so the earlier the disease comes under treatment. That many cases of ulcer of the stomach undoubtedly recover completely without any special treatment is shewn by the frequent discovery after death of scars without any history of the affection during life, others similarly recover by judicious and thorough treatment, whilst others again recover for a time, but sooner or later relapse; others in consequence of some complication suffer from further gastric or abdominal symptoms at a remote date, and lastly, some succumb to the lesion either from inanition, haemorrhage, or perforation. The statistical records available for an estimate of the outlook in gastric ulcer shew that medically this lesion is treated too lightly, and that treatment is often withheld or curtailed short of bringing about its cure. According to Leube's experience 75 per cent of all cases will be cured by four to five weeks' medical treatment according to his method; 21 per cent will be

benefited by a single course of treatment, which should be repeated two or three times before surgical measures are resorted to. Out of 424 cases he found that 74·1 per cent were cured, 21·9 per cent were improved, 1·6 per cent were not relieved, and 2·4 per cent died. According to Lebert the mortality of gastric ulcer is about 10 per cent; about 6½ per cent terminate fatally by perforation into the peritoneal cavity; death from haemorrhage is said to occur in about 3 per cent; in a few death is due to exhaustion or to one of the rarer complications mentioned above. Dr. J. W. Russell, who traced a small number of cases of gastric ulcer which had been treated medically, found—

Death	immediately resulting from the ulcer, in	2·1 per cent	6·4 per cent.
	from intercurrent disease . .	,, 4·3 ,,	
Recovery	having had only one attack .	,, 27·7 ,,	42·6 per cent.
	,, ,, several attacks . .	,, 14·9 ,,	
Remaining on the border-line between recovery and continuance . . .			6·4 per cent.
Still suffering from recurring attacks .		,, 14·9 ,,	44·7 per cent.
,, ,, continuously. . . .		,, 29·8 ,,	

Dr. Bulstrode's statistics reveal equally discouraging results; in 500 cases admitted to the London Hospital from 1897 to 1902 he found 18 per cent fatal, namely, from peritonitis in 10 per cent, from haemorrhage in 2·5 per cent, and from other causes in 5·5 per cent; of these 500 cases it was calculated that at least 42 per cent had previously been under treatment for gastric ulcer, which implies that relapse occurs in 42 per cent. Further, these statistics do not include the cases of deformity, cancer, and other sequels of ulcer admitted to the hospital. In 158 consecutive cases admitted to the London Temperance Hospital the percentage of real cures was less than 25 [Paterson and Rhodes (119)]. In startling contrast with these figures from British sources is Fleiner's experience of 300 cases treated by him medically during ten years; in these there was not a single death, not a single case of severe haemorrhage, nor of uncontrollable vomiting. In 27 cases only was gastro-enterostomy necessary to facilitate the emptying of the stomach.

Treatment.—*Prophylaxis.*—We have seen that gastric ulcer frequently occurs in chlorotic young women. It is well, therefore, in all cases of chlorosis to regulate the diet carefully, and to see that the patient avoids very hot food, coarse food, and food rich in acid and condiments which irritate the mucous membrane of the stomach. Continued pressure on the stomach, such as is produced by tight lacing or by a faulty stooping posture, is also to be avoided (109).

The *treatment of the ulcer* must be directed towards aiding by every possible means the natural reparative powers of the tissues, and so the rapid and sound healing of the lesion. The immediate indications to the attainment of this object, were such a lesion in a visible situation, would manifestly be to reduce the ulcer to its smallest possible dimensions, to maintain it in that state at absolute rest, to protect it from

irritation of all kinds, and to eliminate as far as possible the conditions that interfere with healing. Such are the conditions we must seek to attain in the treatment of ulcer of the stomach, and that the position of the lesion precludes the possibility of controlling the healing process by actual inspection, should be a reason for excessive rather than defective caution. These indications are to be met by rest, not only of the stomach, but also of the body generally. This treatment—the "rest cure"—insisted on as fundamental by Cruveilhier, early recommended by Wilson Fox, Foster, and Williams in England, and in Germany by Ziemssen and Leube, must, if it is to be uniformly successful, be carried out in a thorough and systematic manner. The patient must be absolutely confined to bed for at least two weeks and be kept as quiet as possible. The stomach must be permitted to contract and be maintained in a state of either complete rest by stopping all food by the mouth or of comparative rest by the administration of bland non-irritating food in small quantities at a time, and the healing process contributed to by warm applications to the epigastrium, fomentations, poultices, compresses, or Leube's stomach-capsule. This consists of a tin capsule filled with hot water and so shaped as closely to fit the epigastric region; it has a metallic ring on each side, so that it can be easily fastened, and so may be worn even when the patient is about.

To give the stomach complete rest it is necessary to stop the administration of all food by the mouth; and, since it has been shewn that the injection of nutrient enemas is followed by the secretion of gastric juice, some authors (Pasteur, Sharkey) recommend the plan of giving nothing beyond rectal injections of plain water, 10 to 15 ounces every four or six hours at a temperature of 100° F. By this method, which is continued for seven to ten days (in some cases even as long as three weeks), with perhaps the administration of a little peptonised milk by the mouth towards the end of the week, it is maintained that the necessity for rectal feeding is done away with, that the general condition of the patient remains good, that healing occurs rapidly, that nursing is simplified and the patient is more comfortable. By exclusive rectal feeding, on the other hand, an attempt is made to meet the nutritive requirements of the body and at the same time to maintain the stomach in a state of rest. The beneficial effect of this procedure was shewn by Dr. Donkin, who was the first to treat a large number of cases of gastric ulcer by exclusive rectal feeding. He extended the treatment over as long as twenty-three days and obtained uniformly favourable results. The method, however, possesses certain inherent drawbacks, and is accordingly of limited applicability. It would appear impossible to maintain the nutrition of the body even at rest by rectal feeding owing to the deficient absorption through the mucosa of the colon, so that there is ever the danger of causing general inanition, which itself would militate against the reparative processes in the stomach, and even of establishing a condition of acid intoxication. Again, patients with gastric ulcer may develop parotitis on rectal feeding, a complication against

which the use of antiseptic mouth-washes does not seem to afford any protection (121). Accordingly this method is to be recommended when the vomiting of food is persistent; when the ingestion of food causes great pain; when there is haemorrhage from the stomach; and when perforation has occurred, or appears to be threatening. But even in these cases exclusive rectal feeding rarely need be prolonged beyond a week or a fortnight. The patient takes nothing per mouth except small pieces of ice or small quantities of water to quench the thirst; for the rectal feeding many physicians recommend artificially digested food, such as peptonised milk-gruel, peptone suppositories, and the like: many, however, prefer to give enemas of beef-tea and raw eggs, with a little brandy (in all about 2 or 3 ounces); the peptonised food is more readily absorbed, but the observations of Ewald shew that enemas of eggs act quite as well and are quite as well absorbed; peptonised suppositories sometimes irritate the bowels, or are not well retained, and, unless recently prepared, are sometimes passed out again unaltered. Recent observations have shewn that the following substances are the most freely absorbed and are the most useful constituents of nutrient enemas: pure glucose, powdered peptones (somatose), powdered casein (sanatogen, plasmon, proton), alcohol, and salt. They should be administered in a concentration not greater than 10 per cent of each constituent, the salt excepted, which is usually given in ·5 per cent concentration. The bulk of each enema should not exceed 10 ounces, from 5 to 7 ounces being most useful. Brandenburg recommends enemas of the following composition: dry peptone (somatose) 20 grammes, glucose 20 grammes, salt 1 gramme, water 200 c.cm. Casein may be substituted for the peptone and alcohol may be added up to 10 per cent. Drs. Boyd and Robertson give the following composition: the yolks of two eggs, 30 grammes of pure glucose, 0·5 gramme of salt, pancreatised milk to 300 c.cm. Such an enema is equivalent to about 300 calories, and of these it is estimated that about 125 are absorbed. Before giving the nutritive enema the rectum should be washed out with a cleansing enema of tepid water; the nutrient enemas should be administered not oftener than every four or six hours.

To supplement feeding by this method, the subcutaneous administration of sterilised olive oil has been recommended; half an ounce may be injected under the skin night and morning, which provides an additional 250 calories per day. Sterilised solutions of pure glucose have been similarly employed. But even in the most favourable circumstances by the combination of rectal and subcutaneous feeding it is not possible to provide more than half the caloric requirement of the body.

Following a preliminary period of exclusive rectal feeding, where such is specially indicated or adopted as a routine procedure, feeding by the mouth is gradually established, and for this purpose *milk* has, since the days of Cruveilhier, been universally regarded as the most appropriate food. It is alkaline, it lessens the acidity of the gastric juice; it does not irritate the mucous membrane, either mechanically or chemically; it

does not call forth vigorous peristaltic movements of the stomach, and it does not remain long in the stomach. Not more than 4 to 6 ounces should be taken at once, and in twenty-four hours 3 to 4 pints may be taken (1000-1400 calories). The milk should be boiled, as Reichmann has shewn that it leaves the stomach more rapidly and forms smaller curds than unboiled milk; it may be taken warm or cold; when it is not well borne, it should be given in small quantities, even teaspoonfuls, at a time, or combined with alkalis, as carbonate of soda or lime-water or soda-water, or it may be pancreatised. Occasionally milk from which some of the casein has been removed (fat milk of Gaertner) can be well borne when ordinary milk is intolerable. Or the milk may, prior to administration, be clotted with rennet (peguin milk). Buttermilk, which contains much less fat and sugar than sweet milk, and is therefore less nourishing, is enjoyed by some patients who cannot take sweet milk. Kefir also has been successfully employed in similar cases. In some cases milk in any form cannot be tolerated, and in addition to rectal feeding, we should try freshly expressed beef-juice, weak beef-tea, Liebig's beef extract, or some of the meat preparations, such as Brand's essences or Valentine's beef-juice, Fleiner's meat-jelly, prepared by boiling chicken or beef with calves' feet, or the meat solution of Leube and Rosenthal. In some cases of vomiting, which persisted even in patients fed by nutritive enemas, scraped raw beef, taken at first in very small quantities, has been well borne, has given relief to the pain and checked the vomiting. I have often, therefore, given the scraped raw beef early in the course of gastric ulcer with considerable benefit.

An exclusive milk diet is a starvation diet, as it is impossible for patients to take the 8 to 10 pints a day which are necessary to provide the caloric requirements of the body. Accordingly this food should be supplemented at an early period—about the tenth day—particularly in patients shewing malnutrition or debility, by the addition to it of protein in the form of raw white of egg or whole egg (1 egg = 70 calories), powdered peptones (somatose), or powdered casein (nutrose, sanatogen, plasmon), of fat in the form of cream or butter, and of such foods as Benger's food, Nestlé's food, powdered rice, arrowroot, and sugar preferably in the form of pure glucose, of which some 60 grammes (246 calories) may be given daily in 20 per cent solution.

An exclusive simple milk diet should be continued for ten days; then, for a week or two, the patient may still have the supplemented milk diet. Then he may take bread and milk or milk boiled with a small quantity of sifted flour; he may also take arrowroot and tapioca. After which, if the pain do not return, the patient may pass on to solids, such as boiled brains, fish, chicken, pigeon, sweetbread or tripe; scraped beef and raw eggs may also be given with this dietary. Stimulants had better be avoided altogether, unless perhaps small quantities of claret or light Burgundy be allowed. With this diet, which may be continued for some weeks, the patient often gains weight; and he may be allowed to take gentle exercise. Gradually he may take minced mutton-chops

and small quantities of underdone scraped steak with stale bread and mashed potatoes and cauliflower. Other vegetables, however, with the exception of rice, are still to be forbidden, as are cakes and pastry. Milk and rice pudding, stewed pears, or baked apples may be allowed. The duration of this dietary depends on the condition of the patient: if he feel sufficiently strong he may now follow his occupation, and make gradual additions to his dietary; the more indigestible food-stuffs, such as certain vegetables and pastry, should, however, not be taken at any time. Many are the diet sheets given by various observers, and adopted in various institutions and private hospitals where patients with gastric ulcer are treated (109).

Patients who have suffered from gastric ulcer should be careful in diet, not only for weeks, but for months, and with the return of any such symptoms as pain or vomiting the treatment with rest and strict diet should again be enforced.

In 1901, at the Congress for Internal Medicine, Lenhartz suggested a concentrated egg-albumin diet for the treatment of gastric ulcer, as one more suited to combat the hyperchlorhydria, and reinforce the enfeebled and anaemic state of the patient. This gave very good results in hospital practice. Details of the method are as follows (67): absolute rest in bed for at least four weeks. All mental excitement is to be avoided. An icebag is placed upon the stomach, and kept there almost continuously for two weeks; this prevents flatulent distension, promotes contraction of the stomach, and alleviates pain. On the *first day*, even when haematemesis has occurred, the patient receives from 7 to 10 ounces of iced milk given in spoonfuls, and from two to four beaten eggs within the first twenty-four hours. At the same time, bismuth subnitrate is given three times a day in doses of 30 grains, and is continued for ten days. The eggs are beaten up whole (with a little sugar) and the vessel containing them is placed in a dish containing ice. A little wine may also be added. The amount of milk taken each day is increased by about 3 ounces, and the number of eggs by one, so that at the end of the first week the patient is receiving up to 30 ounces of milk and from six to eight eggs per day. These are continued in the same daily amounts for another week. Some raw minced meat is usually allowed on the sixth day, about one ounce being administered in small quantities at a time. On the following day the amount is doubled, and, later, may be still further increased, if well digested. On the seventh day some well-cooked rice and a few softened Zwieback are allowed. In the third week a mixed diet is resumed. The bowels are not moved during the first week, and in many cases not until the end of the second week, small glycerin injections or warm water enemas being employed; after the third week this procedure is carried out daily if spontaneous evacuation does not occur. The patient is allowed to get up on the twenty-eighth day, and is discharged from the sixth to the tenth week.

Tabulation of Lenhartz's Diet

	Days after last haematemesis.													
	1	2	3	4	5	6	7	8	9	10	11	12	13	14
Eggs*	2	3	4	5	6	7	8	8	8	8	8	8	8	8
Sugar with eggs . grammes	0	0	20	20	30	30	40	40	50	50	50	50	50	50
Milk c.c.	200	300	400	500	600	700	800	900	1000	1000	1000	1000	1000	1000
Raw chopped meat . grammes	0	0	0	0	0	35	70	70	70	70	70	70	70	70
Milk rice	0	0	0	0	0	0	100	100	200	200	300	300	300	300
Zwieback	0	0	0	0	0	0	0	20= 1 piece	2 pieces	2	3	3	4	5
Raw ham	0	0	0	0	0	0	0	0	0	50	50	50	50	50
Butter	0	0	0	0	0	0	0	0	0	20	40	40	40	40
Calories	280	420	637	777	956	1135	1588	1721	2138	2478	2941	2941	3007	3073

* From the first to the seventh day inclusive the eggs are beaten; from the seventh to the fourteenth day inclusive half are beaten and half are cooked.

Among others, Senator has adopted the régime of Lenhartz with certain modifications. He recommends a diet consisting of gelatin, fat, and sugar. Gelatin is easily digested, it possesses a high nutritive value, being a saver of protein, it increases the coagulability of the blood, and thereby helps to stop bleeding. Fat and sugar both possess high nutritive values; the former, in addition, exerts an inhibitory effect on the secretion of hydrochloric acid and a sedative influence on the base of the ulcer. Cases admitted to hospital with recent haematemesis are placed at once on teaspoon doses of a decoction of white gelatin with sugar (gelatin 15 to 20 in 200 to 250 water and 50 elaeosacc. citri) administered every one to two hours, or, in severe cases, every quarter or half-hour. Fresh butter and cream are also given in small quantities frequently repeated, so that, in twenty-four hours, the patient receives 1 ounce of butter and 9 ounces of cream. If butter cannot be taken in its ordinary form, Senator gives it in small fragments, which are frozen hard and sucked by the patient. The cream, which may, if preferred, be mixed with cane sugar, can be whipped to a snow and kept on ice. In this way the patient, immediately after a haematemesis, receives food having a daily value of from 900 to 1000 calories, double the number supplied in the egg and milk diet of Lenhartz. If there be no recurrence of the haematemesis, milk, beaten eggs, and scraped raw meat are added to the dietary, the decoction of gelatin being gradually left off, but at once resumed in the event of any subsequent haemorrhage. Ewald (51), however, is of opinion that the giving of food by the mouth, however bland, immediately after a haemorrhage involves risks from distention of the stomach and displacement of the thrombus, which outweigh the advantages claimed for the Lenhartz method. In such cases he withholds all food by the mouth for two or three days, and resumes oral feeding on the fourth day, beginning with milk (10-15 oz.) and cream (3-4 oz.) in the twenty-four hours; on the ninth day he adds eggs, and from the eleventh day onwards he adds biscuits and gruel.

For the treatment of chronic ulcers with marked dilatation of the stomach I refer to the article on "Dilatation of the Stomach" (p. 548).

Almost as essential as the diet is the strict supervision of the function of the bowels. Constipation is a common accompaniment of the gastric ulcer, and its proper treatment is as important as the dietetic measures. Carlsbad salts, or a saline aperient such as a combination of sulphate of soda and sulphate of magnesia, or some mineral water, such as Carlsbad water, Hunyadi Janos, or Aesculap, are the chief aperients recommended. Carlsbad salts enjoy special favour with Continental observers—one or two teaspoonfuls are given in a tumblerful or half a tumblerful of warm water before breakfast. These salts, apart from their purely aperient effect, are said to act beneficially by diminishing the hyperacidity of the gastric juice, and by increasing the motor power of the stomach (Spitzer, Jaworski). In many cases the good effects of this aperient cannot be doubted; but in some the salts increase the pain and discomfort after meals, and diminish the appetite: in such cases I have found simple enemas and the administration of an infusion of senna pods to be of service. Eight to ten senna pods are added to 10 ounces of boiling water, the water is allowed to cool and to stand for some hours, and then the pods are removed. Generally after the infusion is taken a satisfactory action of the bowels follows in six to eight hours. Rhubarb and cascara sagrada are useful in the more chronic cases.

This "rest cure" in its several degrees may of course be modified to suit individual peculiarities; experience has shewn that it is reliable, and failure is as a rule due to lack of thoroughness in carrying it out.

Medical treatment in many cases may help the dietetic method. The remedies which are here to be specially recommended are bismuth in the form of subnitrate or carbonate, and bicarbonate of soda. Bismuth salts have long been recommended by English writers: the salt may be given either in powder or in suspension; the doses usually given are from 10 to 15 grains. Much larger doses, however, of bismuth are employed by Fleiner, following the recommendation of Kussmaul. One hundred and fifty to three hundred grains of bismuth are suspended in about 8 ounces of water, and the mixture is passed into the stomach, previously washed out by means of the stomach-tube: it is then allowed, by guiding the tube, to remain in the stomach for fifteen minutes, during which time the patient occupies such a position that the bismuth is brought into contact with the ulcerated surface (if the situation of the ulcer can be determined); after this lapse of time the fluid is allowed to run out again through the tube. At first this method is applied daily; after a time, once in two or three days. When the passage of a stomach-tube is contra-indicated the bismuth mixture is to be drunk. The bismuth is supposed to act mechanically by forming a covering which protects the ulcer and facilitates the healing of it.

This treatment, according to Matthes, Fischer, Steitzing, and others, gives most satisfactory results, and appears to bring about a much more speedy healing of the ulcer. Against the method several objections have been urged, such as the danger of using the stomach-tube, or the evil

effects of large doses of bismuth which may set up vomiting and diarrhoea. The occurrence of the latter is very doubtful even when very large doses are employed, and, as Pariser pointed out, neither the stomach-tube nor the maintenance of any special posture is necessary, since it has been shewn by Matthes that the bismuth is rapidly spread in uniform layer over the mucosa of the stomach after its administration. For some years I have given very large doses of bismuth in cases of gastric ulcer—20 to 50 grains three or four times daily: I have never seen any ill effect from these large doses; on the contrary, in most cases the large doses, especially if a small dose of morphine or hydrochlorate of cocaine were added, relieved the epigastric pain very speedily and also quickly stopped the vomiting. I agree with other observers, such as Mathieu, Savelieff, Wegele, in thinking that large doses of bismuth are so well borne and give such good results that one may generally dispense with the irrigation method.

Of the various preparations of bismuth the subnitrate appears to answer best, and it can be given either in powder or in suspension; 10 to 15 grains should be given at first, and the dose may afterwards be increased to 20 and 30 grains or more: Boas prefers the carbonate as being less likely to act on the function of the intestine. The bismuth should be given before meals, and if the pain be very great, or the irritability of the stomach excessive, it may be combined with one-tenth of a grain of morphine or one-fourth of a grain of hydrochlorate of cocaine; if the appetite be deficient, small doses of dilute hydrocyanic acid and tincture of nux vomica may be added to the mixture. Pariser has modified this method by employing in place of bismuth a mixture of chalk 4 parts, talc 4 parts, and magnesia usta 1 part, of which from two to three drachms are given, stirred up in water, before breakfast and again at night three hours after the last meal. The results obtained are equal to those obtained by the bismuth salts, and the mixture possesses the advantages that it does not blacken the stools and so mask any haemorrhage, and by virtue of the magnesia, the proportion of which can be varied to meet the peculiarities of each case, the action of the bowels is regulated. The carbonates of soda, lime, and magnesia have for a long time been given in cases of gastric ulcer. Of these the first—the bicarbonate of soda—is still largely given; some French observers, notably Debove, recommend very large doses (300 to 450 grains) daily; they report very good results, and state that such large doses are very well borne by the patients, who complain only of increased thirst and increased urination; occasionally, however, such large doses produce rather profuse diarrhoea, which can, however, be obviated by giving some carbonate of lime with the bicarbonate of soda.

Nitrate of silver is also an old remedy for gastric ulcer, and I have been in the habit of giving it for years, especially to patients, such as the out-patients of hospitals, who cannot afford to undergo the rest treatment; I have found in many cases that it relieved the pain even better than morphine, and that it gave relief to the other symptoms also.

I have given the nitrate of silver either in the form of pills (one-fourth of a grain to the dose) or in solution (one-fourth of a grain). Boas recommends it very highly in the liquid form, and gives it in small but gradually increasing doses, beginning with half a grain three times daily and going on gradually to one grain. I have given it in more than 200 cases, and have never seen any signs of argyria; it may be given for several weeks and then discontinued for some time.

Treatment of Symptoms and Complications.—Pain.—The application of hot poultices, rest, and regulation of the diet often suffice to relieve the pain; the use of bismuth and nitrate of silver has also been mentioned for the severe paroxysms of pain (gastralgia). Codeia, or better still morphine, either by the mouth or subcutaneously, is indicated: given in the latter form, morphine, according to Leubuscher and Schäfer, and Hitzig, diminishes the secretion of hydrochloric acid in the gastric juice; although the more recent observations of Riegel shew that it has precisely the opposite effect of increasing the acidity of the gastric contents. Atropine, on the other hand, diminishes the secretion of gastric juice and also inhibits the motility and the muscular spasm. Gerhardt also believed atropine to be more efficient in many cases than morphine. The efficacy of belladonna and atropine I have proved in a large number of cases, and now give it as a routine procedure in cases with marked hyperchlorhydria and cramp. In this class of case the administration of olive oil or almond oil recommended by Cohnheim, Walko, and others, in doses of from 1 to 2 ounces, acts beneficially by diminishing the secretion of hydrochloric acid, reducing the pyloric spasm, soothing the base of the ulcer, and subserving nutrition.

Vomiting.—For persistent vomiting abstention from food by the mouth for some days is to be recommended; of drugs drop doses of dilute hydrocyanic acid, or bismuth subnitrate in large doses, with or without cocaine, often act well. Drs. Rolleston and Jex-Blake found that a certain percentage (27·1) of patients with gastric ulcer who were being fed exclusively per rectum, vomited material other than blood; in some this vomiting appeared to be reflex from irritation of the rectal injections, and ceased on the cautious resumption of oral feeding; in others it was apparently a sequence of oral sepsis, as it was cured or ameliorated by removal of bad teeth.

Haematemesis.—The patient is to be kept absolutely at rest and in the recumbent posture (he should not even be allowed to get up to pass urine or faeces), and all food by the mouth should be avoided; if the patient be excessively thirsty this should be relieved by rectal injections of warm water, not exceeding 15 ounces at a time. If the haemorrhage persist, ergot in the form of ergotin should be injected subcutaneously and ice applied to the epigastrium. In profuse haematemesis I have given turpentine with the greatest benefit; it may be administered either in the form of capsules or as an emulsion (two to three teaspoonfuls of turpentine beaten up with the white of one egg). The dose, about twenty to thirty minims, may be repeated after some hours if the

haemorrhage persist. I could cite several cases of haematemesis from ulcer in which the patient was pulseless and blanched to an extreme degree, in which ice, gallic acid, ergotin injections, and other styptics had been tried in vain, and in which the first dose of turpentine completely stopped the haemorrhage. It is well borne.

Adrenalin chloride 1 in 1000 solution may be administered in doses of 10 to 20 m. three times a day. Dr. Hale White recommends Liq. ferri perchlor. in half-drachm doses with an equal quantity of glycerin. High rectal injections of hot water 112°-120° F. repeated several times if necessary, as recommended by Tripier, may also be tried.

If the anaemia produced by the haemorrhage be excessive (the pulse scarcely perceptible, the patient shewing signs of syncope, and the voice becoming feeble), then transfusion or injection into the subcutaneous tissue of one pint or more of normal salt solution may be tried.

I am still of opinion that for some days (three to six) after the last haemorrhage the patient must be fed exclusively by enema, and must keep the recumbent position; after that period the treatment for the healing of the ulcer by rest, liquid food, and hot applications to the epigastrium must be systematically carried out. In cases in which profuse haemorrhages occur from time to time and exhaust the patient, operative interference is called for. This has been successfully done in a number of cases since 1887, when v. Mikulicz performed the pioneer operation for this condition.

Treatment of Perforation of the Ulcer.—Spontaneous recovery from this accident can only occur if the stomach be completely empty when perforation occurs. In such cases a circumscribed peritonitis develops, which eventually leads to the formation of adhesions. If, however, perforation be followed by the escape of stomach contents into the peritoneal cavity, immediate operative interference affords the only hope of recovery; without such interference the accident is fatal in 95 per cent of all cases. The time for operative measures is as soon as the diagnosis of perforation can be made; the later such measures are undertaken the smaller are the chances of recovery, as is shewn in the following table from Messrs. Robson and Moynihan:—

	Total.	Recovered.	Died.	Percentage of Deaths.
Under 12 hours . . .	49	35	14	28·5
From 12 to 24 hours . .	33	12	21	63·6
From 24 to 36 hours . .	16	2	14	87·5
From 36 to 48 hours . .	2	—	2	100·0
Over 48 hours . . .	33	16	17	51·5

In a continuous series of eleven cases operated on within a few hours of rupture (T. Sinclair Kirk, 1905) there was no mortality.

Surgical interference may also be called for in the treatment of chronic indolent ulcers of the stomach which remain unbenefited by a thorough

and systematic course of medical treatment; in such cases the ulcer-bearing area may be excised, or, as is more frequently done, the stomach is ensured comparative rest by the formation of a gastro-enteric fistula. In severe haemorrhage, which recurs despite medical treatment, and also in bleeding from a chronic ulcer—whether slight or severe—surgical measures are called for, and should be undertaken in a quiescent interval, but this should not be waited for to the detriment of the general condition of the patient. In tetany of gastric origin, drainage of the dilated stomach by gastro-enterostomy has proved successful in the hands of Mr. Mayo Robson. In pyloric stenosis, both cicatricial and spasmodic, associated with obstructive dilatation, in hour-glass deformity, in disabling perigastric adhesions, and in perigastric and subphrenic abscess, surgical procedures are of course indicated.

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J. D.

TUMOURS OF THE STOMACH

By W. Hale White, M.D., F.R.C.P.

Primary carcinoma being the most frequent and important of the tumours of the stomach, and the only form of common clinical interest, will be considered first. Towards the end of this article, secondary carcinoma, carcinomatous infiltration from some neighbouring organ, sarcoma, fibroma, fibromyoma, adenoma, and non-malignant villous tumours will be briefly described.

The stomach is a very common seat of primary malignant disease, which is nearly always carcinomatous; no organ in the body is more frequently affected; indeed in nearly half of all the cases of carcinoma the primary seat of disease is in the stomach. I find at Guy's Hospital that in the ten years 1886-96 it caused the death of 60 patients, that is to say, in hospital practice it is responsible for 1·5 per cent of all deaths. Authors are agreed that it is commoner in men than in women; some give the proportion as 3 to 2, others as 2 to 1; Sir C. Perry and Dr. Shaw give the proportion as 5 to 2, and it is important to remember that all their cases were examined after death. It is a disease of the later half of life. Perry and Shaw found the average age at death to be 52·1 years. Of their cases, all examined histologically, the youngest sufferer from carcinoma of the stomach was 32 and the oldest 81. According to Brinton, three-fourths of the cases occur between 40 and 70; between 40 and 50 is, according to Perry and Shaw who analysed 283 cases, the most fatal decade, but that between 50 and 60 is almost as fatal; probably the age at death is a little later now than it was, for in some cases life has been undoubtedly prolonged by short-circuiting. About 2·5 per cent of the cases occur between 20 and 30. The average age at death is a little earlier with women than men. It has been alleged that sometimes cancer of the stomach is met with in young subjects; but it is very probable that some, at least, of these cases were instances of sarcoma, for among Perry and Shaw's series of 48 cases, all examined histologically, no case of cancer occurred in a patient younger than 32; but of four cases of sarcoma, one patient was 15 and another 18 years of age. Dr. Norman Moore, however, has recorded an undoubted case of carcinoma of the stomach in a girl aet. 13, and Osler and M'Crae were only able to find records of 13 cases in the second decade—of these Dr. Moore's was the youngest; under 10 years of age they collected six cases only, and about some of these there was considerable doubt. Probably cancer of the stomach is rarer in negroes than in white people.

Etiology.—We know little of this subject. The only fact that has been said to bear on it is that some cases of gastric carcinoma grow from a simple ulcer. The percentage of cases of ulcer of the stomach followed

by cancer is given as between 5 and 9, but the last is certainly too high. Osler and M'Crae found no evidence that a gastric ulcer was especially liable to become the seat of a carcinoma. Fagge thought otherwise, and Perry and Shaw found simple ulcers in 9 per cent of the gastric carcinomas examined after death, although according to Welch's figures gastric ulcers, either active or healed, are only found in 5 per cent of all autopsies. This appears to shew that there is a slight special liability for gastric ulcers to become carcinomatous. Some authors state that a large proportion of patients with gastric carcinoma have previously suffered from ulcer, but their conclusions depend on clinical histories, and I have elsewhere shewn that many patients supposed to have a gastric ulcer have not (28). In Guy's Hospital Museum there are two specimens of carcinoma beginning in a gastric ulcer; in both, the growth is a spheroidal-celled carcinoma; both patients were women, one aged 39, the other 65. As the pylorus is the narrowest part of the stomach, it is the part most likely to be irritated by the passage of indigestible masses of food; and this may explain the greater prevalence of cancer at the pylorus. Brinton found the growth at the pylorus in 219 out of 360 cases; and Habershon gives the following table, shewing the position of the growth in 79 cases which he examined at Guy's Hospital:—

Pylorus	in 41	Middle	in 4
Lesser Curvature	„ 11	Multiple	„ 1
Cardia	„ 10	Greater Curvature	„ 1
Anterior Wall	„ 5	Cardia and Pylorus	„ 1
General	„ 5		

These figures agree very closely with those given by Osler, and those given by Welch after a study of 1300 cases. Perry and Shaw found the pylorus affected in 70 per cent of the cases.

There is no evidence that trauma, any particular food, or chronic indigestion leads to cancer of the stomach, or that heredity plays any part. Occasionally we see carcinoma in a patient affected with phthisis, but I do not know that this association is especially marked when the cancer grows in the stomach. There is no trustworthy evidence as to whether cancer of the stomach is increasing in frequency.

Morbid Anatomy.—The naked-eye appearances of malignant disease of the stomach vary greatly, and depend chiefly on the extent of the growth, the amount of fibrous tissue in it, and the degree of contraction or ulceration. There may be so much fibrous tissue that the new growth appears exactly like a dense, hard, innocent fibrous tumour, and its true nature is only revealed by an examination of the lymphatic glands. Such tumours are often sharply defined, are frequently limited to the pylorus, shew much hypertrophy of the muscular coats, and yield no juice on scraping. There are all degrees between these and those rapidly growing ulcerated masses which, sloughing, suppurating, or bleeding, destroy all the coats of the stomach beyond recognition, and form such

repulsive specimens. A common intermediate form is a hard, well-defined tumour usually invading the pylorus, having a distinct edge, and extending along the lesser curvature. On section the whitish new growth in the submucous coat stands out in well-marked contrast to the darker hypertrophied muscular layer, especially at the outer part of the tumour; for at the centre the muscle is often destroyed by the carcinomatous tissue, or has several whitish strands of growth running parallel to its muscular fibres. The peritoneal coat is opaque and puckered, and the total thickness of the stomach wall may be half an inch or an inch. If the growth have undergone colloid degeneration, or be a villous tumour, its appearance will be modified accordingly. When the whole of the wall of the stomach is affected, it forms what is known as the india-rubber bottle stomach, in which case the walls are usually much thickened —they may be half an inch thick—and the new tissue is so contracted that the stomach is very small (*vide* p. 439). In a case at Guy's Hospital it was seven inches long, and its cavity was so contracted that it only measured two inches across at its widest part. The growth may adhere to and invade the liver, the pancreas, the spine, the intestine, or the abdominal wall; but adhesions to the liver, intestine, and pancreas are much the commonest, and these organs are often extensively invaded. To complete the picture we must imagine that in most cases there are secondary growths, that fistulous communications sometimes form, and that when the growth is at the pylorus the stomach is generally much hypertrophied and dilated.

Histology.—Sir Cooper Perry and Dr. Shaw have written such a full article, giving an account of the material at my disposal, namely, that in the Museum at Guy's Hospital, that I cannot do better than base my description largely on their paper, especially as Osler and M'Crae consider their classification very convenient. They analysed 48 cases (4 of sarcoma and 44 of carcinoma) of malignant disease of the stomach, and they divide the carcinomas into cylindrical- and spheroidal-celled carcinoma, the latter being three times the commoner. The tumours called by some authors destructive adenoma are included among the cylindrical-celled carcinomas, for in most specimens it is easy to see many transitions between the two forms. Of 11 cylindrical-celled carcinomas, 5 were limited to the pylorus and encircled it; 2 implicated the lesser curvature and cardia; 1 affected the entire organ; 2 implicated the pylorus and a good deal of the stomach besides, and 1 was limited to the posterior wall. Thirty cases of spheroidal-celled carcinoma were described, and the different regions of the stomach were for the most part affected in the same proportion as in cylindrical carcinoma, the pylorus being invaded in about 70 per cent of both classes. The only exceptions concerned growths limited to the cardia, of which there were 3, and all were spheroidal-celled carcinoma; and growths implicating the entire organ, 7 out of 8 of which were spheroidal-celled carcinoma. Age and sex, apparently, had no influence on the form of carcinoma; and no difference could be made out between the two forms as regards the probability of

secondary growths or the organs in which they would occur; the figures given on p. 506 will apply, therefore, to either form.

Colloid degeneration appears to be much more common in spheroidal than in cylindrical-celled carcinoma; for it was observed in 1 out of 12 cases of the cylindrical, and in 9 out of 32 cases of the spheroidal kind. Two of the whole series of 48 cases were malignant villous growths, both cylindrical-celled carcinoma; circumscribed globular or subglobular tumours also usually belong to this variety, but diffuse infiltrating growths, especially when accompanied by contraction, are almost certainly spheroidal carcinoma. Either variety may be medullary or scirrhous, both lie mostly in the submucous coat, but the medullary variety destroys the coats and projects externally more than the scirrhous; and on the whole, medullary cancer is more common at the cardiac than at the pyloric end. Eight (17 per cent) of the cases fall under the heading of india-rubber bottle stomach, the wall being universally indurated and the cavity of the viscus mostly contracted. In these cases the mucous membrane is nearly always smooth, the submucosa is thickened, especially towards the pylorus, the muscular coat is hypertrophied, and the serous coat opaque. Seven of these 8 cases of india-rubber bottle stomach were spheroidal-celled carcinoma, with much fibrous tissue, the amount varying with the degree of contraction of the organ. There may be so much of it that many slides must be examined before any cancer cells are found; but even in cases in which they are so few that no section happens to reveal them, the carcinomatous nature of the tumour is betrayed by an examination of the lymphatic glands. Perry and Shaw believe that all universal fibrous indurations of the stomach not due to swallowing corrosives are caused by hard and slowly growing carcinoma. And, further, a considerable number at any rate of cases of so-called non-malignant scirrhus of the pylorus (fibrous pylorus) not due to corrosive poisoning or the contraction of an ulcer, are really cancerous. Certainly I am disposed to agree strongly with both these propositions. The only exception I know is that in the chronic inflammatory thickening which occurs around the gall-bladder in some cases of gall-stones the pylorus may be very thick and fibrous (compare, however, pp. 437 and 533). Welch was unable to find an authentic case of primary melanotic tumour of the stomach either sarcoma or carcinoma.

Sir C. Perry and Dr. Shaw paid particular attention to the origin of carcinoma restricted to the cardiac end of the stomach. As I have already said, they found 4 specimens, which were all spheroidal-celled carcinoma. But among over 20 cases of oesophageal carcinoma examined, not one was spheroidal; it is highly probable, therefore, that these growths began in the stomach and spread into the oesophagus rather than, as Fagge supposed, that they spread the other way (*vide* p. 346). This is supported by Dr. Fawcett, and is of interest when it is remembered that carcinoma of the stomach never spreads beyond the pylorus to the duodenum (*vide* p. 577).

Symptoms.—These begin very gradually and insidiously, and have

commonly been present some time before the patient consults a doctor and less than eighteen months before he seeks admission to a hospital. He usually says that for the last few months, or it may be longer, he has been troubled with indigestion, one symptom of which is often pain. So common is this mode of onset that gastric carcinoma should always be suspected in any patient who, after the age of 50, becomes chronically dyspeptic for the first time in his life. Many mistakes are made because this rule is forgotten. When the patient is cross-questioned he usually admits that lately he has felt weak, has lost flesh, and has become pale. Still, as the total number of cases of gastric carcinoma is large, it is by no means rare to meet with instances in which it has never been suspected till secondary growths are perceptible, usually in the liver; and then again there are the exceptional cases in which the onset is quite sudden, the patient having previously been in excellent health. At the early stage of the disease there may be nothing specially noteworthy about the dyspeptic symptoms, indeed they may be absent; but the loss of appetite and repugnance to food are usually well marked, and these are important early signs. The tongue is furred, the patient complains of a sense of fulness and heaviness in the gastric region, and often of pain and eructations. (For gases of the stomach see p. 283.)

Pain of some sort or another is almost constant, and is responsible for the drawn look, expressive of much suffering, which the face of a patient suffering from cancer of the stomach usually shews. It is present at some period of the illness in about 90 per cent of the cases. There are three varieties of it, namely, that due to the indigestion, that due to the cancerous growth, and that due to distension; but it is very difficult to separate them. When, however, the pain is principally between the shoulders, and is benefited by taking food, it is probably dyspeptic. The pain characteristic of the cancer itself may be very severe, but speaking generally it is not so extreme as is that of ulcer; it is principally felt in the epigastric region, but it may radiate widely. Except in the early stages it is usually continuous, and may or may not be altered by the ingestion of food; but, independently of this, it often varies in severity from time to time, and may even be paroxysmal. Patients describe it differently, but generally it is either a wearing pain, or it shoots; in the latter case it is agonising, and may then be as severe as that of an ulcer, and it is almost always increased by pressure. That due merely to distension of the stomach is usually relieved when the stomach is emptied either by vomiting or lavage. The pain of a gastric malignant tumour bears no relation to its size, the depth of ulceration, or the rapidity of growth.

The stomach probably gets its sensory nerve supply from the 6th, 7th, 8th, and 9th dorsal segments; the 6th and 7th furnishing the cardia, and the 9th the pylorus. Hence, referred pain from a gastric carcinoma is often felt over those areas of skin which, as Dr. Henry Head has shewn, correspond to these dorsal segments; that is to say, in front from just below the nipple to the umbilicus, and behind from just below the

5th to just below the 12th dorsal spine. Further, the cutaneous tenderness—generally best demonstrated by pressure with the head of a pin, which, if the tenderness be excessive, makes the patient flinch or even cry out—may often be elicited over these areas when the stomach is diseased. Each area has points which are more sensitive to this pressure than the rest of the area. Those for the 6th dorsal area are just under the nipple and at the angle of the scapula; those for the 7th are near the tip of the ensiform cartilage and below the angle of the scapula; those in front, for the 8th and 9th areas, are both in the nipple line; the 9th being on the costal margin, the 8th above it; behind, the 8th tender area is below the 7th, and the 9th below the 8th. When the stomach is diseased these tender spots may be very evident; but cutaneous tenderness is of less value in gastric carcinoma than in other diseases of the stomach, for cases of malignant disease are complicated by the presence of tender areas due to secondary deposits in other organs: moreover, in long-standing and exhausting diseases, pains often radiate far beyond the areas usually associated with the affected organ. Many patients complain of pains in the head, and the scalp should be tested for local tenderness. Dr. Head has shewn that there are painful and tender areas on the scalp which correspond to dorsal cutaneous areas; and they may be the seat of pain and tenderness when either is felt in its corresponding dorsal area. The occipital area corresponds with the 10th dorsal, the parietal with the 9th. The area, which is triangular with its base at the middle line of the head and its apex in front of and above the ear, corresponds to the 8th dorsal; and the temporal area just behind the outer part of the eyebrow corresponds to the 7th.

When these dyspeptic symptoms have lasted some time, the patient begins, in most cases, to complain of nausea, commonly succeeded by vomiting, which, unlike that of most other gastric affections, usually does not completely relieve the pain, although he feels more comfortable after it. This symptom is present in 87 per cent of the cases. It is most troublesome when there is some stenosis of the pylorus; and it bears more relation to the volume of the contents of the stomach than to the ingestion of food. The vomited material is faintly acid, dark brown, often smells very disagreeably, and in exceptional cases stinks horribly, when it has undergone butyric acid fermentation or, in rare cases, putrefaction. Under the microscope pieces of undigested food (which may have been swallowed days or weeks before), crowds of micro-organisms—especially sarcinae, blood-discs fresh and altered, and, in excessively rare cases, cells derived from the growth may be seen. If vomiting is frequent and only small quantities are brought up the growth may be infiltrating the whole stomach and contracting it (india-rubber bottle stomach), and if absent it is more likely to be mural than pyloric. If dysphagia be present the growth is probably at the cardiac orifice, but this symptom may be absent even with a growth in this position.

Much attention has been directed to the kind of acids present in the

gastric contents in cases of gastric carcinoma. Golding-Bird, assistant physician to Guy's Hospital from 1843 to 1854, was the first to make observations, and he shewed that towards the end of life hydrochloric acid gradually disappeared and that organic acids appeared. For many years this was unnoticed, but in 1879 van de Velden also shewed that free hydrochloric acid was absent in the cases of gastric carcinoma which he examined. The vomit itself may be examined, but it is much better to wash out the contents of the stomach with pure water in the morning after the patient has been fasting during the night, and then to give a little simple food that will not cause vomiting. If possible, it should contain a fair amount of protein; thus bread and butter with milk are suitable. An hour or two afterwards the contents may be syphoned out and examined for free hydrochloric and lactic acids. Many tests have been recommended for the detection of free hydrochloric acid in the stomach contents. They are discussed elsewhere in this volume (p. 279). It has been clearly shewn by numerous researches that when a patient is suffering from a carcinoma of the stomach, the secretion of free hydrochloric acid is nearly always greatly diminished, unless the examination be made very early in the disease. The diminution is so great that many authors have said that the acid is entirely absent in about nine-tenths of all cases; but while it is often absent, careful quantitative estimations have shewn that it would be more accurate to say that it is nearly always very considerably reduced in quantity. Lactic acid is frequently present in the gastric contents in cases of carcinoma, but like other organic acids that may be also found, it is only due to the decomposition of the contents of the stomach, and therefore is most often found when pyloric obstruction is present. Free hydrochloric acid may be greatly reduced in conditions other than carcinoma, especially atrophy of the gastric mucous membrane, chronic gastritis, and febrile disorders, although it is impossible to say whether all the diseases which have been said to cause a great diminution really do so, for the statement is often made without the corroboration of a quantitative analysis. Still great reduction or absence is so much commoner in gastric carcinoma than in these conditions that either is of great value in diagnosis, although in a small number, probably under 10 per cent, of the cases of gastric carcinoma free hydrochloric acid is present. Riegel is of opinion that this is sometimes because carcinoma has developed on the site of a previous ulcer, and Osler and M'Crae state that the continued presence of hydrochloric acid, sometimes in large amounts, has been repeatedly shewn in connexion with carcinoma developing in a previous simple ulcer; but, as already pointed out, Osler and M'Crae found no evidence that carcinoma was especially liable to develop on an ulcer, and at any rate the liability must be very small (see pp. 476, 496). The diminution of hydrochloric acid in cases of gastric carcinoma is not only greater than in other conditions, but it often comes on remarkably early in the course of the disease, and it is to be particularly noted that it may be well marked when the gastric growth is small and localised. This led to the suggestion that the

diminution is due to the neutralisation of the acid secretion by some secretion of the growth; but this view was overthrown by the researches of Prof. B. Moore, who, making observations on 17 patients, 3 of whom had carcinoma of the uterus, 3 of the breast, and 5 of the mouth, tongue, or cheek, 2 had sarcoma, and the rest carcinoma in other positions, found that in all the free hydrochloric acid secreted after a test-meal was absent or greatly reduced. The Günzburg test shewed no reaction in 12 out of the 17, and in the remainder the percentage of hydrochloric acid was very small, the highest figure being 0·01455 per cent, the normal as estimated by Moore being 0·1951 per cent. These remarkable results have been confirmed by Dr. Morton Palmer, who found a very great reduction in the percentage of free hydrochloric acid in many patients suffering from carcinoma of organs other than the stomach; thus, in a case of epithelioma of the penis it was reduced to ·00078 per cent. Such results as these make us wonder whether malignant growths wherever situated may not form an internal secretion which, passing into the blood, inhibits the formation of hydrochloric acid by the stomach; if so, this will explain the severe wasting that occurs in malignant disease, for, as Prof. Starling has shewn, the formation of the secretin, which determines the production of pancreatic juice, is dependent upon hydrochloric acid in the gastric juice: if this be so, it suggests to us that all patients suffering from carcinoma wherever situated should take, directly after their food, some hydrochloric acid. Their other digestive secretions might then form naturally, their food might be digested, and they might not waste. It is true that Dr. Morton Palmer found that four other patients in the surgical wards shewed a diminished secretion of hydrochloric acid; they were suffering from pyonephrosis, simple stricture of the rectum, chronic mastitis, and conjunctivitis and keratitis; and B. Moore, W. Alexander, R. E. Kelly, and H. E. Roaf have quite recently confirmed these results. Taking this paper with Moore's first paper, we have a total of 34 cases of carcinoma situated elsewhere than in the stomach, and 20 other cases of patients not suffering from malignant disease. The estimation of free gastric hydrochloric acid shewed that while there was a considerable reduction in the case of the 20 hospital patients who were not suffering from carcinoma, yet there was a still greater reduction in patients with carcinoma. Thus, there was an entire absence in 66·7 per cent of the carcinoma cases, and the amount was above 0·05 per cent in 4 cases only; on the other hand, in the non-malignant hospital patients there is a complete absence in only 25 per cent of the cases, and the amount was above 0·05 per cent in half the cases. The authors point out that these results must be due to an alteration in the pabulum brought to the acid-secreting cells by the circulating fluid, and this alteration may be due either to an organic toxic agent—this is virtually the same suggestion as that given a few lines back, and made before their article was read—or to an alteration in the balance of the inorganic ions of the plasma, as a result of which the concentration in acid or hydrogen ions diminished, so

causing the work on the part of the cell of separating an acid secretion to be increased. For further information on this subject the reader should consult the article by Messrs. Moore, Alexander, Kelly, and Roaf, and also the succeeding one by Moore and Wilson, in which the reaction of the blood in cases of carcinoma is fully discussed. If the suggestion that the absence of free hydrochloric acid is in part the cause of the wasting in cancer be correct, it may be that the wasting of other illnesses in which the free hydrochloric acid is reduced may be due to the diminution of acid.

Free lactic acid is not common in the gastric contents unless the patient be suffering from cancer of the stomach, and in this disease it is often present. Manges found it in 20 out of 21 cases; Ewald found it in 22 out of 24; but Straus found it in 5 only out of 12 non-malignant cases of gastric disease. It is merely the result of the retention and consequent bacterial decomposition of the gastric contents. In doubtful cases it is of the greatest importance to search for hydrochloric and lactic acids.

Microscopic examination of the gastric contents may provide valuable information; fragments of growth may be found especially in the eye of the soft tube used, blood-cells may often be seen when the blood is not visible to the naked eye, and in stained specimens the Oppler-Boas bacillus may be observed. Probably it like lactic acid is only indicative of fermentation.

When the patient comes under treatment the indigestion and vomiting may lessen, and careful diet and rest in bed may even lead to a little gain in weight. Hence he is often buoyed up by false hopes, and the physician may begin to question his diagnosis; but it is a good general rule to be very slow to alter the diagnosis of cancer when it has been reached after a careful survey of the whole case. Any temporary improvement passes away, and the symptoms again begin slowly to increase in severity; the vomiting becomes more frequent, and the wasting is more marked.

In about 35 to 40 per cent of the cases the vomit at some time or another contains blood visible to the naked eye. This is due to the ulceration of the growth, and when fatal there is often ulceration into some large vessel, *e.g.* the splenic artery. If the bleeding be slight, the blood may remain in the stomach long enough to be partially digested; the vomit then looks like coffee-grounds. On the other hand, the haemorrhage may be profuse, and a quantity of bright red blood may be ejected at once; but this is not so common as the "coffee-grounds vomit." Melaena is not very frequent, for it often happens that the pyloric obstruction prevents the blood from reaching the bowel. Gastric haemorrhage from carcinoma of the stomach is rarely fatal.

There is very little to say about the general signs. Considerable enfeeblement is very common, and, being nearly always much more marked than in cases of simple dyspepsia, is often of great help in diagnosis. If the patient live long enough he has the thin, dry, wrinkled skin so

characteristic of cancer; he wastes to a skeleton, and his face is pinched and expressive of great suffering. The loss of flesh in cases of cancer of the stomach is often more rapid than in that due to malignant disease of other organs; for to the wasting due to the disease itself is added that due to the lack of food, the vomiting, and the imperfect digestion. The patient is pale and sallow, rarely there is a slight increase in the number of red cells, but usually they number about 3,000,000 or 4,000,000, and it is very rare for them to fall below 1,500,000; there are often a few poikilocytes, occasionally a few nucleated reds, but no megaloblasts. The haemoglobin generally falls more than the number of red cells, and hence a colour-index of about 0·6 is not uncommon. The leucocytes may be normal or diminished in number, but a slight increase up to say 12,000 or a little more is common, the increase is chiefly among the polymorphonuclear forms. Digestion-leucocytosis is usually absent, but this is not peculiar to, or diagnostic of, gastric carcinoma as was once thought. The patient's temperature is often low, but there may be a mild, usually irregular, degree of pyrexia; the cause of this is unknown; he is excessively weak, the myotatic irritability is increased, there may be oedema of the ankles, the urine sometimes contains a considerable amount of ethereal sulphates, especially indican, peptonuria may appear, and, towards the end, a little albumin and a few casts. Nevertheless it is by no means unusual to be able to diagnose cancer of the stomach by feeling a tumour, and examining the gastric contents before any of these general signs have become striking, and while the patient is in good condition.

Whether the growth produce any signs due to mechanical interference depends entirely on its position. As it is usually at the pylorus, that orifice is often narrowed, and consequently the stomach dilates and becomes over-full. An account of the symptoms of dilatation of the stomach is given in another article (see p. 533). A growth at the pylorus commonly leads to constipation, but sometimes in carcinoma of the stomach there is diarrhoea, probably due to the passage of some of the decomposing contents of the stomach through the pylorus; if so, it indicates that the obstruction is not great. The rectus muscle over the growth is often rigid; in fact, constant rigidity of the right rectus, if other symptoms point in the same direction, supports the diagnosis of carcinoma of the stomach. It is frequently possible to feel a malignant tumour of the stomach, but this clearly depends upon its position, its size, and the thickness and degree of relaxation of the abdominal wall.

To feel a tumour of the stomach to the best advantage the patient should be on his back, with his knees drawn up; he should breathe deeply and keep his mouth open. If, now, the abdominal muscles are not relaxed his attention may be distracted by making him talk. If it is still impossible to form a satisfactory opinion, it may be necessary to give him some chloroform. The examiner's hand should always be warm, and should be moved slowly and evenly over the abdomen. A malignant tumour of the stomach is hard, tender, and often irregular. It varies very much in size, but it slowly increases. Usually a dull note is obtained

over it, but sometimes on deep percussion an obscurely resonant note may be heard. Inflation of the organ with gas may render the tumour more easy to detect. The pylorus can hardly ever be felt in health, so that if this part can be made out by palpation it is pretty certainly diseased. If enlarged it is usually felt under the outer part of the right rectus, or at the right border of it; but although tumours of the pylorus are, as a rule, much more fixed than those in other parts of the stomach, owing to the close attachment of this structure to the liver, yet not infrequently these attachments stretch, and the tumour becomes freely movable over a range of as much, it may be, as three or four inches, so as to suggest the presence of a floating kidney; but it is more usual for tumours of the stomach to become more and more fixed, as in the course of time they form more numerous and denser adhesions. A tumour of the pylorus moves up and down with the liver in respiration, and it is usually impossible, either by palpation or percussion, to separate it from the liver. It must not be forgotten that even an enlarged pylorus is frequently so covered by the liver that it cannot be detected during life. If the growth be on the anterior surface and along the greater curvature, it is more easy to detect, is more mobile under the hand, and alters its position when the stomach is distended with gas; but it does not move so freely with respiration. If, as is sometimes the case, it forms a mass along the greater curvature, we feel a hard, irregular, tender tumour running transversely across the abdomen above the umbilicus. This is very liable to be confounded with a thickened puckered omentum, such as occurs in chronic peritonitis of any form, or with a growth in the colon, or the thickened lower edge of a diseased liver. Tumours limited to the cardia can seldom be felt. The stomach may be distended with carbonic acid gas if the patient swallows first a solution of bicarbonate of soda and then some lemon-juice, and sometimes this may help diagnosis, for we may when the stomach is distended be the better able to tell whether a tumour is in its wall, and also whether the tumour is adherent to surrounding organs; but this mode of investigation often helps very little, and some patients dislike it. If a pyloric tumour move as the result of inflation, the change of position is to the right and a little downwards; if inflation render a tumour previously palpable no longer tangible it is either in the posterior wall of the stomach or behind that organ. A tumour of the stomach is sometimes large enough to form a visible projection of the abdominal wall; and towards the end of the case, when the patient is very wasted, it may sometimes be seen to ascend and descend with respiration, frequently making a considerable excursion. The dilated stomach is often visible, and well-marked peristalsis of it may be seen (for signs of gastric dilatation see p. 540). If the growth lie over the aorta it may be lifted up and down by the pulsations of this vessel. In those cases in which a hard growth infiltrates the whole of the stomach-wall (india-rubber bottle stomach) a hard mass may be felt lying across the abdomen in the epigastric angle, the vomit is small in amount and occurs soon after food, and the organ cannot be inflated.

Complications.—Secondary growths in other organs are very common. On an analysis of 38 cases of malignant disease of the stomach, all examined histologically, collected by Perry and Shaw, no secondary deposits were found after death in 7 cases (18 per cent), a percentage obtained by other writers. They occurred in one organ in 15 cases; in two organs in 8 cases; in three organs in 6 cases; in four organs in 1 case, and in eight organs in 1 case. All authors agree that the lymphatic glands are most frequently affected, being infiltrated in a little over a third of all cases; the liver is affected in about a third, the peritoneum in about a fifth, the lungs in about an eighth, and the pleura in about a tenth; but growths may be found anywhere in the body. These secondary deposits frequently cause important symptoms; thus, the affection of the liver, or of the glands in the portal fissure, often leads to fatal jaundice, or the implication of the peritoneum, helped by pressure of some mass on the portal vein, leads to ascites. Secondary growths often cause death from bronchitis and pneumonia, and when the pleura is affected there may be pleural effusion. The left supraclavicular glands should always be examined, for they are sometimes enlarged by secondary infiltration, and this may be so in cases in which it would be otherwise impossible to come to a correct diagnosis. The enlargement has been found after death to be due to direct spread up the thoracic duct, but it has been suggested that sometimes infection takes place by other lymphatic channels through the diaphragm. Occasionally the left axillary glands are affected. The navel, too, may be implicated by palpable secondary growths; usually, but not always, the peritoneum then has secondary nodules in it.

In cancer of the stomach, as in all wasting diseases, thrombosis is prone to occur. It is most frequently seen in the left saphena or in the left femoral vein. The wasting may induce a fatty liver and a fatty heart, and death may be due to cardiac failure; or the patient may become so thin that, if the nursing be inefficient, a bed-sore may form and hasten his end.

The growth in the stomach is, by the time death occurs, ulcerated in three-quarters of the cases, and by its ulceration it may open a large artery; death may then result with profuse arterial haematemesis, but this is rare. Guy's Hospital Museum contains a specimen in which the splenic artery was thus laid open. Ulceration into the spleen has caused fatal haematemesis from that organ. Out of Perry and Shaw's 306 cases bleeding caused death in 11, in 6 of these there was haematemesis, and in the other 5 the autopsy shewed that gastric haemorrhage had proved fatal without any vomiting of blood. Abscesses not due to actual perforation may arise in connexion with the growth. Often these are only found at the necropsy; but there may be a collection of pus in the subphrenic region large enough to be detected during life. In some cases rigors occur; these are usually due to suppuration, but it is said that both rigors and pyrexia may occur in its absence. Not infrequently the patient appears too weak to shew any signs of suppura-

tive processes. The drowsiness and the delirium which have been observed may perhaps be due to toxic absorption from the foul contents of the stomach; indeed, we do not know how far such a cause may be concerned in the production of general symptoms.

Perforation occurs in about 7 per cent of the cases: it happened in 20 out of Perry and Shaw's 306 cases; other authors do not put the percentage quite so high, but it must be remembered that it is often overlooked in the absence of an autopsy. In 13 of the 20 cases perforation took place into the general peritoneal cavity; if the patient be very weak and exhausted when this happens there may be no evidence during life of this event. In the remaining 7 cases perforation took place among adhesions, and a local abscess followed; in two there was an abscess between the pylorus and the liver, in one the abscess extended from the liver to the spleen, in one it was in the lesser sac of the peritoneum, in one over the lesser curvature of the stomach, in one between the abdominal wall and the stomach, and in one the position is not stated. Such abscesses may lead to a communication between the stomach and colon. Thus faecal vomiting may be set up with a horrible taste in the mouth and odour of the breath; but if the stomach be constantly distended and full of fluid, there may be no passage of the contents of the colon into it. In about a third of the cases of gastro-colic fistula undigested food may be observed in the faeces; whenever food appears soon in the faeces in an undigested state the condition is termed lientery, but this may occur when there is no fistulous communication. Other fistulas are extremely rare; but there may be a communication between the stomach and duodenum, or a gastro-cutaneous fistula may form. Adhesions in connexion with the growth are very common, being found in about two-thirds of the cases examined after death.

Termination.—It is clear that several of these complications may kill the patient; but not uncommonly he appears to die slowly from sheer weakness, lying during the last few days of his life quite still, almost pulseless, and with hardly any respiratory movements. I have often noticed that, as Fagge points out, many of the symptoms diminish towards the end; the pain is less, and the vomiting stops; but I do not know that this remission is peculiar to cancer. Near the end of any exhausting disease all the functions of the body, even the conducting power of the nerves, are depressed; consequently no pain is felt, and reflex acts fail. I have seen cases of perforation in enteric fever and in cancer of the caecum in which during life there had been no signs to lead me to suspect what had happened. Welch points out that sometimes patients with gastric carcinoma die comatose. Carcinoma of the stomach is especially rapid in young subjects.

Latent Cases.—In any large series from the post-mortem room there will be a small number of cases in which cancer of the stomach is found after death, although unsuspected during life. Eight out of Osler and M'Crae's 150 cases were latent. As Perry and Shaw point out, the latent cancers of the stomach may be divided into two groups

—(1) those who die from cancer of the stomach without its being suspected during life, and (2) those who die with cancer of the stomach without its being suspected during life. With regard to this group Welch remarks that it is rare to find cancer of the stomach in an apparently healthy man dying of an accident; this is true, for out of 21,260 post-mortem examinations Sir C. Perry and Dr. Shaw only found 7 such cases. All the patients died of some malady other than cancer of the stomach, and there were no symptoms pointing to cancer of the stomach; the case of a woman from whose history nothing could be learnt suggesting cancer of the stomach, although when she died from a strangulated hernia a gastric carcinoma extending from the pylorus along almost the whole of both walls for five inches, is of great interest. Among the 306 cases of cancer of the stomach occurring in the 21,260 autopsies they found 13 examples of the first group of latent cases. Mistakes with regard to these cases can only be avoided by great care (see Diagnosis).

Diagnosis.—It may be difficult to distinguish between ulcer and carcinoma of the stomach; but the subjects of ulcer are younger than sufferers from carcinoma. They commonly give a history of previous similar attacks; or at any rate they say they have before suffered from indigestion; but malignant disease of the stomach often occurs in persons who have never had indigestion. The pain of ulcer is made worse by food, and is relieved by vomiting; in cancer this is less common. Wasting is a less marked symptom in ulcer, and the patients do not so often complain of loss of appetite and repugnance to food. The absence of hydrochloric acid and the presence of lactic acid and sarcinae are strongly in favour of cancer; and the presence of secondary growths is of course conclusive. Prof. Osler quotes the case of a man in whom a small nodule in the anterior abdominal wall was associated with gastric symptoms of uncertain origin. It was excised, found to be carcinoma, and the diagnosis of gastric carcinoma thus arrived at proved to be correct. Profuse haemorrhage and bright redness of the blood are in favour of ulcer, and the patients affected with this malady rarely have the appearance of those suffering from cancer. Lastly, a duration of more than eighteen months is much in favour of ulcer. The most difficult cases are those in which an ulcer has by its adhesions formed a tumour in the region of the pylorus, and the difficulty is enhanced if this also leads to dilatation of the stomach, and I have known induration about the gall-bladder induced by gall-stones implicate the pylorus, and by causing a tumour and gastric dilatation, lead to a diagnosis of cancer of the stomach.

In the early stages the diagnosis between cancer and chronic catarrhal gastritis is often impossible; but, as already remarked, when symptoms of indigestion come on for the first time after the age of fifty they are nearly always due to cancer; but under thirty hardly ever. After what has been said about carcinoma, the reader will easily be able to perceive the signs which, in a later stage of cancerous disease, will prevent a mistake.

From a practical point of view it is often a difficult question whether a patient who is obviously ill and anaemic is suffering from pulmonary tuberculosis, pernicious anaemia, or from a latent carcinoma of the stomach. No useful purpose will be served by going over the points of diagnosis, for each case must be judged on its own evidence. The best way to avoid a mistake is to remember the possibility of it. Great care may be necessary to determine whether a tumour in the abdomen springs from the stomach or not. If it is in the pyloric region, it may be attributed to the liver or gall-bladder; or it may be that from stretching of the attachment of the stomach it is mistaken for a movable kidney. The pylorus may in such cases be felt as a tumour below the umbilicus, and may be moved three or four inches with the hand. An excellent case in point is figured by Prof. Osler. Great attention must be paid to all the symptoms of the individual case. If the stomach be dilated, or gas can be felt on pressure to bubble through the tumour, it is in the pylorus. Growths in the body of the stomach often form a hard solid mass running transversely across the abdomen, so that they are very difficult to tell from the edge of the liver, a thickened puckered omentum, a tumour of the colon, or a faecal accumulation. In such cases it is of the greatest possible importance to observe the precise position of the tumour, its extent, and its relation to the edge of the liver; and to distinguish the gastric resonance from that due to the colon. Inflation of the stomach may help us much; and it is often wise not to give too positive an opinion until the bowels have been well opened by an enema. In rare cases it has been difficult to distinguish between an enlarged spleen and a tumour at the cardiac end of the stomach. If a patient be made to swallow two or more drachms of bismuth carbonate in a little milk or mucilage while he sits in a chair, the metal may be seen with the *x*-rays to fall to the lowest part of the stomach, and thus we learn whether it is dilated. In many cases no tumour can be felt during life, and the organ is not dilated, but even then a diagnosis may often be made by the other signs. The most difficult cases are those in which extensive secondary growths in other organs divert attention from the stomach; indeed, sometimes there are no gastric symptoms, and although it may be recognised that the growths we discovered are secondary, we have no clue as to the primary focus.

Prognosis.—A malignant tumour of the stomach must sooner or later kill, unless it be successfully removed. A few exceptional cases have been recorded which appear to shew that life may be prolonged for even four years after the onset of symptoms, but it is always difficult to be sure that the early symptoms were due to the growth, and it may safely be said that life is rarely prolonged more than eighteen months, or at the outside two years, after the patient is first led to consult a doctor for the symptoms of this disease. The duration of the greater number of cases is much less than eighteen months, and often is less than a year; indeed, the average duration is only nine months, and with very many it is much less. A great deal depends upon the rate of growth of the tumour and

the rapidity with which secondary growths form. Under careful dieting and rest in bed improvement may occur for a time. This is particularly likely if the sufferer is a hospital patient who has not had good food and rest outside the hospital, and forgetfulness of this has often led to mistakes in diagnosis.

Treatment.—Except in very rare cases this can only be palliative. In most instances subcutaneous injections of morphine are necessary sooner or later, and they should be given whenever pain is severe. It is cruel to withhold them from a man who is suffering agony and has only a few months to live. Much relief is often afforded by daily lavage, and by not allowing much fluid to be drunk, and benefit especially follows when the stomach is dilated or when there is vomiting. When the loss of appetite is a prominent symptom, benefit may follow the use of an acid bitter mixture; indeed we have already seen reason for believing that acids will almost always be helpful. For example, ten minims each of dilute nitro-hydrochloric acid and tincture of nux vomica, with some compound tincture of gentian, or spiritus armoraciae compositus, given before meals, will help the appetite and compensate the deficient secretion of acid. A glass of sherry and bitters before a meal is of use in stimulating the secretion of acid. As a rule, it is difficult to prevent fermentation by drugs; but carbolic acid, naphthol, mineral acids, and salicylic acid may be tried, although most of these, if given in doses sufficient to prevent fermentation, irritate the stomach; on the whole, perhaps, salicylic acid is the best. Careful attention must be paid to the diet. The food should be given in small quantities at frequent intervals, and generally the dieting resolves itself into giving what the patient finds he can best digest. While on the one hand the food should be simple and capable of easy digestion, such as peptonised milk, on the other hand it should not be such as will easily undergo fermentation; small quantities of meat and jelly are therefore useful, but in different cases the physician has to deal with such varying degrees of failure to digest and liability to decomposition that no detailed rules can be laid down. Constipation frequently requires treatment; some pleasant tasting purgative, such as the aromatic syrup of cascara, is best, and should be given shortly after lavage. Often enemas or glycerin suppositories are necessary.

Two surgical operations call for discussion. First there is the question of the removal of the growth. Unfortunately by the time malignant disease has produced symptoms sufficiently marked for the sufferer to consult a doctor, it is usually so advanced that excision is either impossible or undesirable on account of metastases. This is undoubtedly true of hospital patients and those seen in consulting practice. Not long ago Mr. Dunn operated upon the most favourable case I have seen; the growth was completely excised and many secondary affected glands were removed, but it is clear they were not all taken away, for although the patient improved for a time he died within eight months from secondary deposits. Although the counsel of perfection is

to excise very early and even to perform an exploratory operation with a view of seeing if a growth be present, yet in practice the percentage of cases suitable for excision is small. If the growth is tangible it is sure either to be too large or adherent for excision, or the secondary growths are so numerous as to make excision inadvisable. It is true that many cases of apparently successful removal of the growth have been recorded, but it is quite likely that some of these were examples of inflammatory thickening in connexion with an innocent ulcer, for it is often quite impossible at an operation to tell this from malignant growth. The most successful and most recent series of cases is that recorded by Mayo, 18·1 per cent of his cases were alive and well three years after excision of the growth. Other operators have not been so fortunate. Excision is most likely to be successful when the patient is operated on early, when all the glands and lymphatics are removed for a large area round the growth, and when the patient is not too young or too old. The best operation is to excise the growth and sew up the cut ends of the intestine and stomach and do a gastro-enterostomy. Secondly, there are the operations with the object of overcoming pyloric obstruction; they are as follow:—the stomach has been opened, and through the opening parts of the cancer have been scraped away with a curette or some suitable instrument. This method should never be adopted; the relief is very transient and the bleeding may be dangerous. Some surgeons have simply excised the pylorus solely to relieve obstruction, but of 66 cases quoted by Sir F. Treves, 50 died soon after the operation, and in all the remainder there was speedy recurrence. We are not surprised, therefore, that the operation has been discarded. Messrs. Cheyne and Burghard well say, "It must never be forgotten that pylorectomy is a severe operation which the surgeon is not justified in performing unless the lesion be so limited that there is a reasonable prospect of a cure." Unfortunately it rarely is so limited. In a few cases the duodenum or jejunum has been brought to the surface of an abdominal wound, and stitched there, and when adhesion has taken place the bowel has been opened and the patient fed through the opening; but this operation too has been discarded, as the patients have soon died after it. A much better operation is to open the abdomen and form a fistulous opening between the stomach and some prominent coil of the jejunum. If it should be decided that the patient is suffering mainly from the pyloric obstruction, and that he is in such a condition that an operation is justifiable, then gastro-jejunostomy should be performed; by its means considerable suffering may be avoided, and the life of the patient probably prolonged for a few weeks or even months. I saw with Mr. G. F. Glinn, a lady aged 91 suffering from a dilated stomach due to pyloric carcinoma, and at our request Mr. F. C. Wallis performed a gastro-jejunostomy. The relief was very marked, the sickness stopped, the patient gained weight, and was able to take a walk. It has already been mentioned that even in the cases in which the pylorus has been excised it is well to do a gastro-jejunostomy as well. Loreta's operation and pyloroplasty are not justifiable for malignant stricture of the pylorus.

Sarcoma of the Stomach. — I have incidentally mentioned that Sir C. Perry and Dr. Lauriston Shaw, out of 50 cases of primary malignant disease of the stomach, found that the growth was sarcomatous in 4. In all it was of the round-celled variety. The new growth was most extensive in the submucous coat, but the mucous, serous, and muscular layers were affected. Dr. Shaw has published a fifth case, and, analysing these 5, and 3 published in the *Pathological Society's Transactions*, he points out that the mucous membrane is rarely invaded in sarcoma, consequently the growth rarely ulcerates and haematemesis is exceptional. The muscular coat is extensively destroyed, the growth rarely contracts, hence dilatation of the stomach and vomiting are not often present. The tumour grows rapidly, is sharply defined, and attains a great size; it may extend past the pyloric ring to the duodenum. The patients are often young adults. It will be noticed that there are many points of contrast between sarcoma and carcinoma of the stomach. Sarcoma is not specially confined to one sex. Among Perry and Shaw's 4 cases 2 were males and 2 were females. The ages were 15, 18, 38, and 67. As the youngest patient with carcinoma was 32, it is justifiable to suspect sarcoma when we have all the symptoms of malignant disease of the stomach in a patient under 30 years of age. In three of the cases secondary growths were found in other organs. Recently Dr. Salaman has investigated the subject very fully from a histological standpoint; he recognises four varieties, namely, lymphosarcoma, round-celled sarcoma, polypoid fibro- and myxo-sarcomas of the stomach, and sarcoma of the peritoneal glands directly invading the stomach. The classification of all these tumours is notoriously very difficult, and probably several histologists would not accept Salaman's classification. In the first two groups the age of 14 patients is given, and 6 were under 25 years of age, thus confirming the statement just made that sarcoma of the stomach often occurs at a much younger age than carcinoma. One of Salaman's own cases belonged to the first group. The growth in these is not limited to the stomach for its origin; it may start in glands as well as the stomach, or it may start from the pharynx and spread down. These growths, when once they have attained a footing in the intestinal canal, have a great tendency to spread along it, and these cases are those described by some authors as lymphadenoma. In the second group, the round-celled sarcoma, the growth arises in the stomach wall itself, does not spread to adjacent parts, and secondary enlargement of the glands is very late. Dr. Salaman considers that it is limited by the pylorus, but other authors do not agree with this. The cases in the third group are clinically quite distinct from the others, for the tumour is nearly always very large, weighing it may be 14 lbs., and often it is pendulous, hanging in the peritoneal cavity from its attachment to the stomach, so that during life it would probably not be recognised as a gastric tumour. From the few figures available it appears that this variety is commoner in women than in men, and is rarely seen under 50 years of age. Secondary growths are very uncommon. The

fourth group is obviously quite distinct from the others, the mass of growth is usually enormous, there is nothing noteworthy about the age and sex, the disease is particularly malignant, many organs are affected, and there is extensive ulceration of the stomach. The glands just above the left clavicle may be enlarged. Dr. Salaman's article contains an excellent bibliography.

Tuberculosis of the Stomach.—This is excessively rare. Tuberculous ulcers are described on p. 446. A tuberculous tumour arising in the muscular coat of the stomach has been described by van Wart, but is a pathological curiosity and probably unique.

Fibroma of the Stomach.—The older writers described a fibroma of the pylorus; but probably their cases were either slow-growing carcinoma with much fibrous stroma, or the pylorus was thickened either from within as a result of a cicatrising ulcer, or from without as a result of inflammation around the gall-bladder and due to gall-stones. I have seen an instance of this last mistake.

Fibromyoma of the Stomach.—In very rare instances these innocent tumours, which may be of any size up to a pigeon's egg, are found projecting into the stomach. They consist of unstriped muscle and fibrous tissue; the mucous membrane over them is intact, and so far as we know they are harmless. Sometimes they are pedunculated, when they form one variety of polypus. Two or three may be present in the same patient.

Adenoma and Papilloma of the Stomach.—Occasionally small, white, translucent non-malignant tumours, composed of tubular structures like normal glands, are found in the stomach. Papillomas are either wart-like or polypoid overgrowths of the mucous membrane; they are very closely related to adenoma. Villous tumours of the stomach are usually malignant; but they may be innocent, and consist of mucous membrane supported on delicate bands of fibrous tissue. Small lipomas may occasionally be seen in the submucous coat.

Lymphadenoma of the Stomach.—This is very rare. Dr. Newton Pitt has collected several cases. In the intestine the lymphoid growth may occur either in the submucous coat, in which case large flat tumours, the mucous membrane of which is not ulcerated, project into the lumen of the bowel; or it may take place in the muscular coat, when the mucous membrane ulcerates and the calibre of the bowel is diminished. Probably the first variety only is met with in the stomach. An increase of lymphoid tissue is always found in other parts of the body, as, for example, in a case recorded by Carrington, of Hodgkin's disease implicating the stomach. It has already been pointed out that Dr. Salaman classes these cases among the sarcomas as lymphosarcoma (see p. 512).

Secondary Growths in the Stomach.—These are rare. Judging by our experience at Guy's, and by other recorded cases, probably about 6 or 7 per cent of malignant growths in the stomach are secondary. The primary growth may be anywhere; the breast is the most common seat. Welch collected 37 cases of secondary carcinoma of the stomach, 17 were secondary to cancer of the breast, 8 to cancer of the oesophagus, 3 to cancer of the mouth and nose, and the remainder to other parts. I have known an epithelioma of the stomach to be secondary to epithelioma of the oesophagus, and there is in Guy's Hospital Museum a specimen of secondary melanotic sarcoma of the stomach. The symptoms do not differ from those of primary disease of the organ; but often those of the primary growth overshadow them, and the patient dies before the gastric symptoms are very manifest.

Foreign Bodies.—Occasionally lunatics and others swallow hair, fibre, or other foreign substances, which slowly aggregate into a mass that may easily be mistaken for a tumour of the stomach.

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CONGENITAL HYPERTROPHY OF THE PYLORUS

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Synonyms.—*Congenital hypertrophic stenosis of the pylorus; congenital gastric spasm.*

Definition.—A gastric disorder of early infancy in which spasm and hypertrophy of the pylorus cause a greater or less degree of obstruction to the passage of food from the stomach to the intestine.

History.—The association of some thickening of the pylorus with symptoms of pyloric obstruction in infancy was reported as early as 1788 by Hezekiah Beardsley, whose report has recently been brought to light by Professor Osler. Between that year and 1888, only two other instances were recorded (Williamson, Dawosky); it was not until 1896 that characteristic signs and symptoms were observed by Dr. J. Thomson of Edinburgh, and by Finkelstein in Germany. Before that time the condition had been merely a pathological curiosity unrecognised during life; its clinical diagnosis has now become easy and its treatment often completely successful. It has also become clear that the condition is by no means so rare as was formerly supposed; more than a hundred cases have been recorded since 1896, and single observers connected with hospitals for children have seen as many as twenty and thirty cases within ten years. Operative treatment was attempted in New York in 1898, and in the following year a recovery after gastro-enterostomy was reported by Abel. Since that time many successes with operation have been recorded, but it has also been shewn by several observers that the condition is amenable to medical treatment.

Etiology.—*Age.*—It has been generally assumed that this disorder is dependent upon some congenital fault either of function or of development. Whilst this seems probable in some if not in all cases, it has not been proved, and it is noteworthy that it is quite exceptional for the symptoms to date from birth. Usually the infant has been perfectly well for some days or weeks after birth; out of 25 cases observed by the present writer, in which the date of onset of symptoms (vomiting) was noted, only 1 began on the day of birth, 4 in the first week, 1 in the second, 7 in the third, 5 in the fourth, 4 in the fifth, 2 in the sixth, and 1 in the seventh. In Sarvonat's recent monograph the time of onset is recorded in 82 cases collected from medical literature; 9 are stated to have shewn symptoms from the day of birth, 6 others in the first week, 67 shewed their earliest symptoms after the end of the first week, 18 in the second week, 17 in the third, 17 in the fourth, 3 in the fifth, 7 in the sixth, and 5 in the eighth. These dates of onset are probably approximate in some cases, for it may be difficult to ascertain exactly

when the symptoms began; the vomiting, which is usually the earliest symptom, has often been regarded at first as the normal regurgitation of infancy.

Sex.—Males would seem to be much more liable than females. Out of 27 cases under my observation, 23 were boys, only 4 were girls.

Family Incidence.—Instances have been recorded of the occurrence of this disorder in more than one child in a family; but this would seem to be rare. It has appeared to me that first-born infants were affected more often than others: out of 25 cases in which this point was noted, 9 were first-born.

Pathology and Morbid Anatomy.—The most striking feature in the morbid anatomy is the thickening of the wall of the stomach and the pylorus. The pylorus, the thickening of which varies considerably in degree, feels unduly hard and on section its lumen is seen to be more or less diminished; this depends in part upon the folding of the mucous membrane brought about by the contraction of the pylorus. The increased size of the pylorus is seen, even with the naked eye, to be due chiefly to overgrowth of the muscular wall, and by the microscope is shewn to depend mainly on increased thickness of the circular muscular coat, the longitudinal layer being only slightly increased. The folding of the mucous membrane gives an appearance in some sections of excess of submucous connective tissue, which is probably apparent and not real. The thickening of the stomach wall, which is more marked near the pylorus than at the cardiac end, is also due to increase of muscular tissue. In some cases the œsophagus has also shewn some thickening of its muscular wall, especially near the cardiac orifice: and the wall of the duodenum in the immediate neighbourhood of the pylorus was also slightly thickened in one of my cases.

Anatomically therefore the condition is purely an excess of muscular tissue. The origin of this excess is uncertain, but attempts to explain it have been made in two different ways. According to one view the excess of muscle is due to a primary developmental fault, an excessive formation of muscle, a true developmental hyperplasia. In favour of this it is stated (Dent quoted by Cautley [6]) that some abnormal thickening of the pylorus has once been observed in a seven-months fœtus, and that it would be impossible for hypertrophy to occur as the result of excessive action within the few weeks which intervene between birth and the occurrence of the clinical signs of hypertrophy. According to others the increase in the muscular wall of the pylorus is a true hypertrophy, the result of excessive action, and this excessive action is attributed to spasm, which may occur in utero or may perhaps begin in some cases after birth. Dr. J. Thomson has suggested that there is some congenital instability of co-ordination which leads to a failure in the normal relation between relaxation of the pyloric sphincter and contraction of the stomach, so that the pylorus contracts instead of relaxing when the stomach is attempting to drive the food into the intestine. In this way an antagonistic action arises between the stomach and the pylorus

which leads to hypertrophy of both, and in the stomach causes almost always a considerable degree of dilatation.

Whether the excess of muscular tissue be due partly or entirely to a developmental hyperplasia, or be the result of increased work, as Dr. Thomson suggests, it seems clear from clinical observation that the obstruction is not a passive one due to any permanent narrowing of lumen. The variation in the degree of obstruction on different days as shewn by the complete absence of vomiting, sometimes for many hours or even for a whole day, without any corresponding accumulation of feeds in the stomach: the absence of any constant relation between the degree of thickening of the pylorus and the degree of obstruction: and the complete and permanent recovery which has several times followed medical measures, such as lavage, in marked and characteristic cases, seem to prove that the obstruction is due, in part at least, to muscular spasm.

Symptoms.—Stated briefly the clinical manifestations of hypertrophy of the pylorus in infants are chronic vomiting, constipation, and wasting, associated with two characteristic signs, visible and excessive peristalsis of the stomach, and palpable thickening of the pylorus.

The first symptom to attract attention is usually the vomiting, and this shews certain characteristic features. The contents of the stomach are ejected more forcibly than is usual in other conditions, so that in most cases the vomit on some occasions comes through the nostrils, and is often ejected to a distance of two or three feet from the mouth. Moreover, after the obstruction has persisted for some time, the amount of the vomited material is greater than the feed last taken, shewing that at least part of another feed has been unable to pass the pylorus. The vomiting, unlike that due to other gastric disorders of infancy, often occurs in spite of correct feeding, in most cases, indeed, the infant is being carefully fed at the breast when the symptoms first appear. It is peculiar also in responding only transiently, if at all, to ordinary dietetic measures; the history often shews that with each change of food there has been some decrease of vomiting for two or three days, after which the vomiting has become as troublesome as before. The vomiting varies much in frequency and degree in different cases, and in the same case at different times. At first, perhaps, the vomiting is only once or twice a day, then it often becomes more frequent, occurring after almost every feed; but when, from the prolonged obstruction, the stomach has become much dilated it may become large enough to allow several feeds to accumulate in it before vomiting occurs, so that the stomach contents may be ejected only once or twice in the twenty-four hours. The vomited matter in the first few days or weeks after the onset of symptoms consists of partially digested food only, but as the stomach becomes dilated, and the mucous membrane irritated by the frequent accumulation of feeds, some degree of catarrh results, and the vomit contains much mucus, sometimes even a trace of blood. There is no bile in the vomit, and there would seem to be no constant excess of acid in the gastric secretion.

Constipation is almost invariably a marked feature of the disorder, and dates usually from the onset of the vomiting. This association is of some importance in diagnosis, for in vomiting due to other gastric disorders there is commonly looseness of the bowels with offensive, green, or otherwise abnormal stools, whereas with hypertrophy of the pylorus in infants the stools, apart from their constipated character, are usually normal. The urine is diminished in quantity, as might be expected, when so little fluid is passing through the pylorus.

Wasting is a prominent sign, and its rapidity is in proportion to the degree of obstruction. In some cases, three or four ounces only are lost per week, in others nearly a pound per week. As emaciation progresses some generalised oedema is apt to occur, especially in the limbs, as is common with marasmus from any cause in infancy. The temperature also, which in the early stage of the disorder is unaffected, becomes subnormal, and when exhaustion is extreme convulsions are apt to occur.



FIG. 16.—Congenital hypertrophy of the pylorus: bulging wave of gastric peristalsis seen rising in the left hypochondrium. (From photograph of case recorded by Drs. Fisher and Neild.)

More characteristic than any of these symptoms are the two signs upon which certainty of diagnosis depends, namely, visible and excessive peristalsis of the stomach and a palpable thickening of the pylorus. The peristalsis is seen as a wave passing across the epigastrium from left to right. A swelling, varying in size from half a walnut to half a tangerine orange, is seen rising up from under the left costal margin whence it travels slowly towards the right side of the abdomen, and before this swelling has yet faded away in the right hypochondrium, another is already appearing on the left side, and sometimes even a third, so that three bulging swellings may be seen at one time travelling slowly one after the other across the abdomen. The position of the peristaltic wave varies according to the degree of dilatation of the stomach; usually it is limited to the epigastrium, but in some cases it travels downwards to the right below the umbilicus into the lumbar region.

The thickening of the pylorus is to be felt usually deep in the right hypochondrium, about two inches below the costal margin, and about one or one and a half inch to the right of the middle line. The fingers should

be pressed deeply backwards towards the right side of the bodies of the vertebrae, and in some cases in which the pylorus is much overhung by the liver, the fingers must be pushed upwards as well as deeply backwards before the tumour can be felt. When the stomach is much dilated the pylorus may be displaced downwards and to the right, so that it is actually in the right lumbar region, and in exceptional cases it is forced forward during peristalsis so as to lie just under the abdominal wall. Both the visible peristalsis and the palpable thickening of the pylorus are to be detected only at intervals, so that repeated examination may be necessary. As they are most readily detected during or just after a meal, it is well to have the infant fed at the time of examination. The pylorus is usually palpable only during the visible peristalsis of the stomach, and at this time can be felt to harden intermittently under the fingers so that at one moment it is easily felt, at the next it cannot be made out at all. If properly sought, the pylorus can probably be felt in all cases; certainly the clinical diagnosis rests on probabilities only in the absence of this pyloric tumour.

Diagnosis.—This disorder is mistaken most often for simple digestive disturbance, and when one kind of food after another has been tried and the infant still vomits, it is regarded as marasmus or gastric catarrh. The association of chronic vomiting with constipation in an infant under four months old, especially if the infant was being reared at the breast at the time of onset of symptoms, should always arouse a suspicion that hypertrophy of the pylorus may be present. In the vomiting due to difficulty of digestion or improper feeding, the bowels are often loose and the stools are usually unhealthy, whereas with hypertrophy of the pylorus not only is there constipation, but the stools are often normal except for their constipated character. But the diagnosis can only be made with certainty by the detection of the two characteristic signs—visible and excessive peristalsis of the stomach and palpable thickening of the pylorus. Peristalsis of the stomach is sometimes just visible on careful inspection in infants immediately after a meal in cases in which there has been chronic vomiting apparently from dyspepsia, or even in infants wasted from any cause; but the peristalsis in these cases can only just be detected, whereas with hypertrophy of the pylorus it is easily visible from a distance of several yards, and has sometimes been noticed by the mother or nurse as something extraordinary before the child comes under medical observation. Congenital stenosis of the duodenum has given rise to symptoms resembling those of pyloric hypertrophy, but there is no thickening of the pylorus to be felt, and the dilated upper part of the duodenum may be detected by palpation: this condition, however, is so extremely rare that it hardly forms a practical difficulty in diagnosis.

Some writers have supposed that a condition of pyloric spasm should be recognised as distinct from pyloric hypertrophy in infants; the symptoms are stated to be similar; but the two characteristic signs are absent or peristalsis, if present, is extremely slight only. It seems

possible that such a condition may exist, and, if so, it may be a mild degree of the same spasmodic affection which in more severe cases is called congenital hypertrophy of the pylorus; but this latter name should be limited to those cases in which the peristalsis is of the excessive degree described above, and in which the thickened pylorus can be felt.

Prognosis. Pyloric hypertrophy in infants is an extremely grave disorder: left to itself, the obstruction and resulting starvation cause progressive wasting until death occurs, usually from exhaustion or convulsions, seldom later than the end of the fourth month. If the condition be recognised and suitably treated recovery may occur, and seems to be permanent and complete so far as present experience allows us to judge: several cases have been watched for three or four years after cessation of symptoms and have shewn no tendency to relapse whether treatment has been medical or surgical. Of my series of 27 cases, 24 occurred since the disorder has been brought within range of treatment: in one of these the subsequent course is unknown: of the remaining 23 cases, 13 recovered completely, one other was gaining weight steadily ten weeks after operation, and appeared to have recovered completely when it fell ill with bronchitis or bronchopneumonia and died. In this series, therefore, at least 50 per cent recovered. Of the 14 cases which recovered [including the one which died subsequently of bronchitis], 8 were treated by operation (forcible dilatation), and 6 by medical measures only. Of the 9 cases which died, 3 were treated by operation (forcible dilatation), two others were already so ill when the diagnosis was first made that they died within three days, and one other was under conditions in which no treatment could be carried out efficiently and the child died within a few days; the remaining three were treated without success by lavage.

Treatment.—It was supposed at one time that this disorder was not amenable to treatment of any kind; later, the success of surgery made it seem probable that operation gave the only hope of recovery. Recently it has been shewn that surgical measures are not always necessary, a certain proportion of these cases recover completely with medical treatment alone. Success has followed medical measures of various kinds. Dietetic measures alone have succeeded only rarely; feeding with very small quantities, such as one or two drachms, of raw meat juice, whey, or veal broth for several days, and then slowly increasing the quantities, has given good results; feeding with a teaspoonful of hot water by the mouth and saline injections by the rectum for one or two days, and then giving only teaspoonful feeds of peptonised milk by the mouth and continuing the saline enemas by rectum, was successful in one case (Harper). Feeding by the nasal tube for several weeks was followed by recovery in one instance (Batten). The most useful, however, of medical measures is lavage. Five cases under my observation recovered completely with prolonged use of lavage, in two others the weight had been rising steadily under this treatment before operation was done. It is important that it

should be realised that recovery is very slow; the lavage should be done twice daily for two or three weeks, and then once daily for several weeks longer; in most cases it has been necessary to continue lavage for at least three months. The stomach is washed out with a weak solution of sodium bicarbonate, two grains to the ounce, introduced through a No. 14 soft rubber catheter, or a small oesophageal tube; the fluid should be at a temperature of 100° F. The feeding should be with small quantities and of such a kind as to leave little or no solid curd in the stomach; sherry-whey in feeds of half an ounce may be used, alternating with feeds of veal-broth. If the infant can be kept upon breast-milk this should be continued in conjunction with the lavage; but if the breast-milk sucked from the breast cause vomiting, it should be drawn off with a pump and given with a spoon or bottle so that the amount of each feed can be regulated, and only about half an ounce or an ounce given every hour.

The question whether operation is necessary must be decided by the result of medical treatment, particularly by the effect upon the infant's weight. The weight should be taken every alternate day; and a continuous loss at two or three weighings, even though the vomiting may have diminished or ceased, is generally an indication that operation is necessary.

The operative measures which have been tried are forcible dilatation of the pylorus (Loreta's operation), gastro-enterostomy, pyloroplasty, and pylorectomy. So far as can be judged from statistics the most successful of these methods would appear to be forcible dilatation of the pylorus; in 11 of my cases treated by this method there were 8 recoveries; according to the records collected by Sarvonat, Loreta's operation shewed 7 recoveries out of 10 cases (his figures include 2 of my successful cases); pyloroplasty is next in success, 8 recoveries out of 16 cases; gastro-enterostomy shewed 11 recoveries out of 26 cases; while pylorectomy was fatal in the single case in which it was tried.

Any one of these methods is necessarily a very serious procedure in an infant only a few weeks or months old. It is to be remembered also that although the operation completely stops the vomiting there is often extreme difficulty in assimilation afterwards, and a liability to diarrhoea, so that death may occur some weeks after operation from this cause or from marasmus.

It seems clear that sometimes operation offers the only prospect of recovery, but there is nothing to distinguish such cases from those which are amenable to medical treatment; the two characteristic signs have been as marked in some which have recovered with lavage or even with the dietetic measures described above, as in others which failed to respond to any medical treatment; it is therefore only right to give thorough trial to these milder measures before concluding that operation is necessary; but delay must not be prolonged if there is still loss of weight, for this would place the infant in a worse position to stand any surgical procedure.

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DILATATION OF THE STOMACH

By Professor T. Clifford Allbutt, M.D., F.R.S.

Synonym.—*Gastrectasis*

Subject.—We are said to be in error if we make "a disease" of dilatation of the stomach, and indeed it is a pathological event which may occur in the course of several diseases. But, as chapters and essays and lectures are devoted to this event, we may assume that it is one so important as to claim independent consideration. Under the name of dilatation of the stomach we shall not include the capacious stomach of gross feeders, and of those persons, labourers and others, who live not unhealthily on bulky, copious, or ill-masticated food: by dilatation we mean a volume of the organ which is excessive in proportion to its ordinary work; and, again, after Rosenbach, we may distinguish between relative and positive insufficiency of the stomach. Certain tribes of men live upon an earth which is rich in organic matter, and among certain coolies this depraved habit is said to be on the increase; the stomach of these men must be indeed voluminous. Among ourselves Trousseau reports 60-80 lbs. of food as the daily intake of a gross feeder; and cottiers who live chiefly upon potatoes are said to possess stomachs capable of containing large quantities of this vegetable. Riegel, by inflation and by soundings after digestion, demonstrated that a stomach, otherwise normal, may yet be of extraordinary capacity—a

condition which has received such names as megastria or megalogastria. Thus, with chemical and motor functions unimpaired, the stomach may descend to the navel, or even a trifle beyond it; and, if the upper border be in place, enteroptosis and "vertical stomach" must be excluded from the diagnosis. Such colossal stomachs, however, must be of rare occurrence, and in fat people are very hard to demonstrate. Dwellers in hot climates live upon bulky carbohydrates—some of them, such as peas, being slow of digestion; but, as in those climates not much fuel is required for the bodily heat, the quantities eaten at one time need not be large.

If, then, the work of the organ be persistently increased, and its nutrition be normal, the stomach, like the other hollow viscera, will strengthen itself accordingly—unless, indeed, the increase be too rapid, or overwhelmingly great. If the work be increased—as, for instance, by a moderate degree of pyloric stenosis—but the bulk of the contents not increased by overfeeding or by fermentation, the volume of the organ may preserve its mean capacity: it may, indeed, be diminished; in rare cases it is enormously diminished, so as to present the form of the so-called "leather-bottle stomach" [*vide* art. "Cirrhosis of Stomach," p. 437].

When the food is delayed in the stomach the viscus is usually, but not always, distended. Food may not stagnate in a big stomach; it may digest slowly in a small one. The result of washing out six hours after a meal should be the basis of our diagnosis, not the measurement of the cubic capacity of the organ, which indeed during life is impossible. And were it possible, there is no standard stomach for a given size of body; such an outline as Fig. 25, p. 862, must be taken as a mean of many observations.

In some cases the contents of the stomach, whatever their source, increase so rapidly, or the activity of the organ falls so fast, or the onward passage of the food is so suddenly arrested, that work is put more or less in abeyance almost at once; the distension cannot be compensated. These cases are called "Acute Dilatation." In other cases, in which excessive demands upon the functions of the stomach come more gradually, the muscular coat may increase in greater or less degree with the capacity; according to the circumstances of the particular case. Extreme instances may be taken from the big quasi-normal stomach of Riegel, and from cases of enlargement due to a narrowing of the pyloric channel (pyloric stenosis). If the pylorus be constricted gradually, the stomach may attain to a huge volume—to a capacity of seven or eight pints or more; but as it is unable duly to empty itself, and by the delay of its contents suffers injury both in motor and secretory power, it cannot retain its normal qualities as a merely big stomach may do. Why in some cases of overwork the stomach thickens rather than dilates is unknown. In the thickening fibrosis may be the chief element, or a very slow carcinomatous permeation; if writhings visible through the integuments are to be taken as sufficient evidence of a vigorous muscular coat, the "leather-bottle stomach" may be a very active one.

Causation.—Duplay père, to whom the first systematic description of the symptoms of dilatation of the stomach is attributed by the French school, who did indeed anticipate Kussmaul in many of the chief points of the clinical description, divided the causes of the disorder into seven kinds: (i.) narrowing of the pylorus; (ii.) abnormal adhesions of the stomach; (iii.) destruction of the muscular fibres; (iv.) induration around the pylorus; (v.) atrophy of the muscular coat; (vi.) hydatid of the stomach; (vii.) palsy of the stomach. This division, as our present knowledge tells us, is both confused and imperfect. Our better understanding of the malady and of its treatment dates from Kussmaul's interesting paper of the year 1869, when washing out with the pump

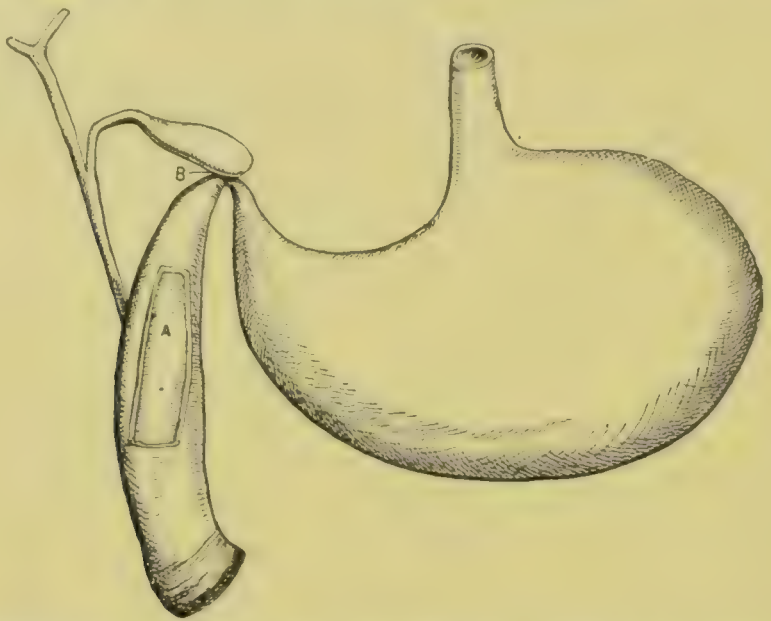


FIG. 17.—Diagram of stomach in which pyloric obstruction was produced by kink caused by adhesion to gall-bladder. [By permission of Mr. Page of Newcastle-on-Tyne, and the editor of the *British Medical Journal*.]

was first recommended—a practice improved soon afterwards by the introduction of the siphon. In 1875 Penzoldt summed up the work on the subject from the date of Kussmaul's essay up to the time of his own; and his treatise contains also a summary of the history of the disease. The number of publications on the subject since the date of Penzoldt's essay are innumerable, at any rate in this place. Penzoldt attributes the clinical distinction between atonic and stenotic gastrectasis to Johann Peter Frank.

The causes of dilatation of the stomach are conveniently separable into these chief classes: those of (A) Atonic distension,[1] more or less persistent, often without descent of the viscus; and of (B) Static dilatation, usually obstructive in origin, with compensatory hypertrophy of the

[1] The name of Myasthenia gastrica has been given by the French school to this condition, but it is unwise to endow subordinate events with imposing titles, and thus to raise them to the apparent dignity of diseases.

muscular coat, and also generally with more or less displacement. In (B) the seat of the obstruction is most frequently about the pylorus; less often in the duodenum (4), rarely in the main cavity of the stomach itself. The pylorus may be thickened by disease benignant or malignant: it may be narrowed by the puckering of a cicatrix; by adhesions without, such as may arise from the irritation of gall-stones or other disease of neighbouring structures; by torsion due to dislocation, or by flexure of a deformed or dislocated stomach at its pyloric extremity (Fig. 17). In (A) the causes of enfeeblement, chiefly of the motor functions, are: (α) A sudden and overwhelming discharge of fluid into the cavity under some influence unknown to us, with arrest of its efflux; as in certain cases of a very acute and perilous kind described by Fagge and other authors under the head of Acute Dilatation. (β) Toxic causes, as in acute rheumatic fever, pulmonary tuberculosis, pneumonia, influenza, enteric fever, infective endocarditis, septicaemia, and other infections. (γ) Improper diet or drink—relative gastric insufficiency, often associated with catarrh, as in chronic alcoholism. (δ) Some weakness in the organ itself, either primary or due to some general failure of health, as in chlorosis, for example, convalescence from acute disease, or neurasthenia (neurasthenia may be no infrequent cause, but as Steele and Francine say, "it is not easy to make a diagnosis of neurasthenia in a brief acquaintance with a walking patient"), whereby its chemical and motor functions, or both of them, are insufficient—positive gastric insufficiency. The gastric distension of portal congestion—as in heart disease—may be included here. (ε) Not infrequently some temporary influence, causing either a neuroparetic state of the wall or a spasm of the pylorus, which can be illustrated but not very easily defined or explained. (ζ) Possibly, but not very probably, an over-activity in the gland cells which secrete the hydrochloric acid of the gastric juice (hyperchlorhydria).

Many of these causes need little elucidation: as regards (ε) Sir Lauder Brunton speaks of a dilatation of the fundus of the stomach in some cases of migraine, during the attacks, and attributes it to spasmodic closure of the pylorus, consequent upon irritation of the vagus nerve; hyperchlorhydria also frequently concurs with such irritation, and sets up the spasm. Mangelsdorf verified Brunton's observation in a large proportion of cases of migraine, and found a like extension in epilepsy. Korn has described a case of intermittent distension of the stomach, which he surmised might be due to a latent ulcer setting up spasm of the pylorus. But a paretic or inhibitory explanation seems in some cases more probable, as in Knoll's note of a sudden and considerable distension of the stomach in a case of severe contusion by which the organ itself was not injured. In a patient of my own, subject to prostrating attacks of left intercostal neuralgia, large extensions of the stomach often appeared during an attack, but not always. In the quiet intervals, of some weeks or months, the stomach was usually normal, and treatment addressed to the stomach did nothing for the neuralgia. Sir W. Bennett's case of the

cure of a dilated stomach by the release of a minute strangulated omental hernia of old standing may have had a like interpretation. I have observed rapid extension of the stomach in more than one case of acute abdominal obstruction, but the toxic explanation is here more likely than the neuroparetic, as in these cases much relief is given by lavage; thus dilatation of the stomach is apt to follow surgical operations upon the abdomen, and to give rise to alarming symptoms, which likewise may be relieved by lavage (p. 550). The distended stomachs in cases of mental stress and worry and other depressing mental conditions may be mentioned here. Such a state of stomach quickly arose in two patients of my own after severe domestic calamities; one case was in 1902 and the other in 1907. A like consequence of excessive athletics in a young and delicate adult I saw some time ago with Dr. Christian Simpson.

Simple distension is often very insidious, especially in the kinds (β) and (δ), whereby convalescence from acute disease may be grievously protracted. Obstructive dilatation is chiefly a disease of adults, and usually of adults in middle life; if it appear in persons over fifty years of age its occurrence will arouse suspicion of malignant disease, especially if there be continuous emaciation and defects of hydrochloric acid; though happily even these symptoms are not always conclusive. The acuter dilatations are frequently seen in young persons, in children, and even in infants: it is not infrequent, for instance, in rickets; but, as pointed out by Mons. Comby, Prof. Hamilton, and myself, it occurs by no means exclusively in this malady. The pot-belly of rickety children is caused, in part at least, by dilatation of the bowels with undigested food, especially starchy food; moreover, in these children the external muscles of the abdomen are abnormally feeble. Both in adults and children dilatation may come on quickly and be considerable in degree, and yet the ultimate prognosis may not be unfavourable: the dimensions, however, never attain those of the larger degrees of mechanical dilatation, nor is the stomach as a whole much displaced. It must be remembered that the normal stomach has times of distension, perhaps of immoderate distension; but the note of normality is that in such cases the organ has not lost the power of recovering itself, and soon does so by vomiting or digestion. From the moment that distension is not followed by such resilience morbid dilatation has begun; stress has led to strain, feebleness to perversion of function; food is delayed in the cavity, and the walls begin to suffer. Temporary dilatation, then, and even temporary insufficiency of the stomach, as of the heart, no doubt occur frequently in the run of life, and in most cases without injury. A distinguished living physician tells me that he has suffered thus three or four times, under the effects of overwork and many dinners; but now and then, when stress is too great or too continuous or the organ is failing in tone, the temporary becomes a persistent disorder. If again, as in Fagge's class of Acute Dilatation (α), the stress be sudden and enormous, the results may be instantly perilous, and indeed fatal.

Atonic distension of the stomach rarely proceeds to definite or persistent

dilatation. It may be that if the appetite is small the stomach is not loaded. Both chemical and motor functions may fluctuate widely between excess and defect, for some authors speak of an abnormally swift digestion in the presence of hydrochloric acid; or a lienteric diarrhoea may set in. Notwithstanding, speaking generally, dilatation is the result rather of motor, positive or relative, than of secretory failure.

No doubt a feeble stomach, not increased in volume, and a dilated stomach, may stand in a like defective relation to an ordinary mass of food; still that the volume of a hollow viscus is a very important factor in its work needs no formal proof. Von Noorden, indeed, discusses under one head all cases in which the stomach does not duly pass on the food into the bowel, whether the state be one of atony or of stenosis. In all such cases dilatation is apt to occur: the ordinary rule is that atony means dilatation, though the converse is by no means a rule.

The falling of the abdominal viscera, noted by Morgagni and others down to Virchow, but now associated with the name of Glénard, is described elsewhere [art. "Visceroptosis"]. If gastroptosis be considerable, pyloric or duodenal kinking may lead to great dilatation; and even if the cavity be not greatly extended, much displacement usually delays the food, and promotes gastric insufficiency.

It is generally believed that catarrh of the organ is the commonest starting-point of atonic dilatation; but in this estimate the frequency of the extension as a complication or sequel of acute disease may not be sufficiently recognised. It is easy during grave illness or during the term of convalescence to attribute debility and dyspepsia to "mere weakness"; it is our business, however, to measure and define where we can, and in a very large number of patients, whether adults or children, in lingering and irregular convalescence after infectious diseases, acute rheumatic fever, enteric, pneumonia, phthisis, and the like, we may detect dilatation of the stomach. In these cases catarrh or imperfect secretion of gastric juice rather than atony or poisoning of the muscular coat may, of course, be the primary local term; as in the pneumococcic gastritis (35A); and often no doubt catarrh and muscular atony are concerned together. However this may be, the result remains that in retarded convalescence dilatation is often present; and that, if a more rapid amendment is to be obtained, treatment of this sequel is necessary. Atony of the stomach leads to more or less delay of its contents, to fermentation, and to secondary catarrh. In putting down dilatation as a disease of mature life—of persons of either sex between forty and fifty years of age—attention has been too exclusively fixed upon cases of obstructive origin, and cases due to more general causes overlooked. When due to these more general causes the malady is as common in the young as in the elderly.

Bouchard suggests that in some persons there may be a specific and even a hereditary feebleness of the muscular fibre of the stomach; that the organ does not contract vigorously between meals, so that the food lies in it, and offers a favourable soil for the microbes of fermentations—

especially if the hydrochloric acid be deficient. Whether then, in dilatation, the chronic catarrh be antecedent, consequent, or absent, the contents of the stomach are relatively or positively too much for it; and this state of things is the worse the weaker the muscular coat. On the other hand, let these causes be what they may—be they the gorging of food and liquids, wholesome or unwholesome, or lowered vitality due to previous illness, or otherwise, — in any or all of these circumstances it is rare to find a dilatation comparable to that of mechanical obstruction; in extreme cases of stenosis the stomach may reach the pubes, in atony never. Indeed, in atony usually the viscus is not dropped, and the area of distension is chiefly an enlargement of the vault of the fundus. Nevertheless, the physician who neglects the factor of dilatation because the stomach is not so blown out, or washy, or dislocated, as to force itself upon his notice, has an imperfect comprehension of his case, and an imperfect hold upon the means of cure; yet not long ago a very eminent London physician said to me that of dilatation of the stomach, except in obstruction, he knew nothing. "Nervous dyspepsia"—apart from the grosser dietetic errors, anaemia, the acute diseases, and the catarrh which plays in and out with these causes—is not, in my experience, a frequent or direct cause of important or persistent dilatation. [*Vide* "Neuroses of the Stomach," p. 396.]

Sudden acute dilatation of the stomach ("Gastroplegia") may occur after a debauch, but the stomach on rare occasions, and under no such insult, may be suddenly overwhelmed and death may swiftly ensue. The two cases, originally published by Fagge under the title of "Acute Gastric Distension," are the classical cases of the kind; but many more have been reported of late years. Both those cases occurred in men, the one being thirty and the other twenty years of age. The symptoms and signs were similar in both, and in cases subsequently recorded; the symptoms may be summed up as pain in the stomach, anuria, and collapse—vomiting is frequent but not constant; the signs are those of a hugely distended stomach charged with fluid. In Fagge's second case the stomach was emptied by the pump, but in spite of this measure it refilled, and death followed in a few hours. In this case a sloughing abscess existed behind the duodenum. On necropsy the mucous membrane was not examined in either, but in both the stomach, when emptied, returned to its normal dimensions. Fagge was disposed to attribute to the stomach itself that fluid which was found in quantity so diluvial. The late Sir William Broadbent told me of such a case of acute dilatation under his care from which eight pints of fluid were removed by the siphon, but no sooner was this volume of fluid removed than the stomach began to refill, and was rapidly expanded again to its former dimensions. Wilson Fox discussed this kind of sudden dilatation, and referred to Humby and Miller's cases which were marked by sudden invasion, vomiting, and death in a few days. Vomiting, when present, occurs at the outset, but as the distension becomes extreme this palliative effort ceases: yet even in Humby and

Miller's cases the stomach, on removal from the body, shrank back to something like its natural size, shewing only white striae, like lineae gravidarum, upon its surface. Similar cases have been reported also by Andral, Peebles, Erdmann, Kundrat, Oser, Boas (8), Connor. Pepper and Stengel suggest that the immediate cause is spasm of the pylorus due to irritation by the contents of the stomach; but Dr. Ewart and others, with more probability, attribute it (in most cases at any rate) to a throttling of the duodenum; and Connor and Thomson in their careful studies of the subject hold a like if not identical opinion. Whether the dilatation or the obstruction comes first seems uncertain, obstruction may be the first step; and this may be by an ensnaring of the lower end of the duodenum between the root of the mesentery, which crosses in front of it, and the vertebral column (*vide* p. 768). Others are of opinion that distension is the first event, the obstructions being produced by the weight of this on the part of the duodenum which crosses the third and ascends by the side of the second lumbar vertebra to the jejunum (13).

Distensions of the stomach, as ordinarily seen, are a much less destructive affair. In debilitating or toxic diseases, or again in chronic non-febrile diseases such as diabetes, in which amelioration is often grievously delayed by it, distension of the stomach may appear with some suddenness; or, if more gradual in its approach, be overlooked in those earlier stages which are concealed by the primary malady. In volume, however, it never approaches that of the fulminating or tightly obstructive cases. Frequently, I may repeat, there is something more than mere atony about it; in rheumatic fever and pulmonary tuberculosis the toxic state of the blood, causing myositis or neuritis, with or without gastritis, may intensify if it do not set up the mischief, as, apart from pericarditis or endocarditis, the heart may in like manner be enfeebled.

On the many debilitating causes which may bring about this atony it is unnecessary to enlarge. Bouchard said that in 80 per cent of chlorotic women the stomach is dilated, but this is an exaggerated estimate.

Dilatation of the stomach not infrequently arises from the ingestion of large quantities of fluid. We see it in heavy drinkers of beer, and in persons who have abandoned themselves to the use of aerated waters, or to the copious tea-drinking not uncommon in the more temperate class of miners, engine-drivers, stokers, and other dusty, thirsty labourers. Unfortunately these workers also consume with their tea a large quantity of carbohydrates; they leave home in the morning after a breakfast of hot tea, bread and butter and cakes, and carry in their pouches more bread and butter and a can of tea, on which they live until the return home, which, in the case of railway men, may be at a very uncertain hour. Thus the stomach, wind-blown rather than overworked, does not hypertrophy after the fashion of the stomach of the over-feeder, whose table is well spread, and whose diet is as varied as his digestion is vigorous and his food nutritious, but tends rather to ballooning. The big, well-fed stomachs,

when dyspepsia drives their owners to the physician, are soon reduced by proper treatment; but the thin, inflated, and ill-nourished organs do not contain within themselves the same faculty of repair. Moreover, the stomach of the glutton often makes more than ordinary quantities of gastric juice; but distension of the walls of an ill-fed stomach, flattening out the glandular structures, impoverishes them; and the food finds, not an increased measure of solvent, but a deficiency. Thus digestion is delayed, flatulency increases more and more, the stomach gives way more and more, and a vicious circle is established. An excessive bulk of food or fluid, then, tends to dilate the stomach: if the food be generous the dilatation is compensated by hypertrophy, and the organ can pull itself together; but if the food be poor and windy the warm imprisoned gases act upon an organ the natural elasticity of which is impaired: in such cases, as I often used to observe in coal-miners, the consequences may be irremediable.

It is said that hyperchlorhydria is a cause of dilatation, but on slender grounds, unless it be by way of pyloric spasm; for the activity of fermentation would be hindered by a persistently high mineral acidity. It is when in the flaccid stomach the food is delayed that easier occasion is given to the agents of fermentation. Thus initial feebleness of muscle and gastric juice leads to delay of the chyme, which falls the more readily into fermentation; and the gaseous products of fermentation, distending walls already lacking in tone, handicap them more and more in the race of peristalsis. The stomach is thus caught in the vicious circle we have noted. If the bulky and sweet farinaceous puddings, too often supplied to convalescents, be consumed in relatively large quantities, things go from worse to worse; the stomach, like a dilated heart, may be stretched beyond ready recovery, and, as Cohnheim says, the patient has "now a vinegar, now a gas factory in his body." There is no superstition more tenacious of life than that which prescribes carbohydrates to all dyspeptics as "so digestible."

Distension due to or associated with portal engorgement is very apt to occur in these forms of cardiac disease; the stomach does not reach to great dimensions, but it is a very frequent and troublesome addition to the disorders more directly cardiac. For example: A woman of about fifty once came under my care for "Heart Disease." I found, indeed, the rhythm of the organ as irregular as it well could be. The stomach was ballooned up to the fourth rib, and extended to the posterior axillary line. She had been nursed and kept almost entirely in bed for three months. Without altering the other conditions in any way we took her off the invalid diet of pap and tea and the like, and put her upon a drier diet of tender meats with less of starchy matter or cellulose, and reduced the tea; in a fortnight her heart had become nearly regular, as regular as after so long a bad habit it was likely to become, and she got so far well that I lost sight of her. The heart was degenerate and dilated, but the flatulent and distended stomach was a great aggravation

of her disorder. A dilated and flatulent stomach may thus disturb, and gravely disturb, even a normal heart; when the heart like the stomach is weak and dilated also, the disturbance is at once more distressing, more persistent, and more mischievous. Probably as the venous pressure in the abdomen rises, the stomach suffers more and more, and yields more and more, whereby the embarrassment of the heart is multiplied.

Spasmodic asthma is grievously aggravated even by moderate gastric extensions; and it is said that such an extension may set up asthma in susceptible persons to whom it might otherwise have been unknown.

Primary gastric catarrh, by reducing the value of the gastric secretion and promoting fermentation, is a potent means of setting up distensions.

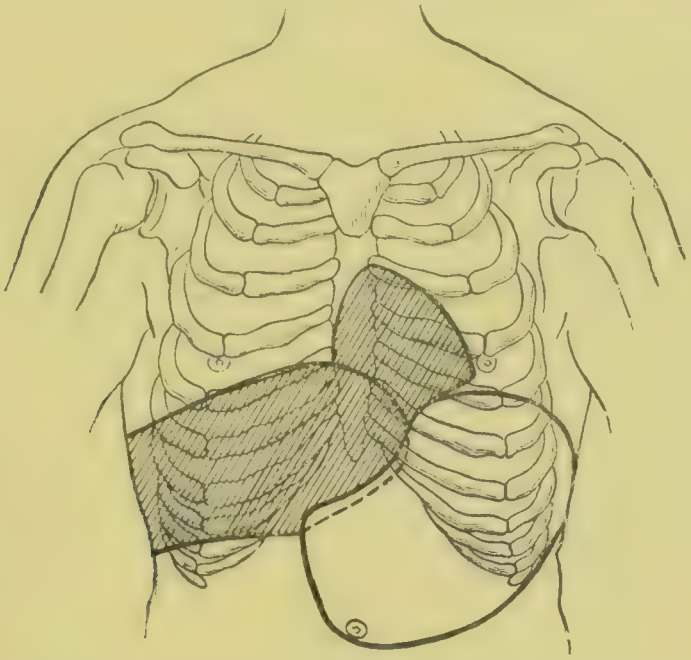


FIG. 18.—Outline of stomach measuring 17·5 cm. vertical, and 22 cm. transverse. From a man aet. 50. Atonic and obstructive dilatation, due to a cicatrix in the anterior wall and adhesions surrounding the pylorus. (Pepper and Stengel.)

The contents of the catarrhal stomach tend to alkalinity, and thus to the harbourage and cultivation of bacteria; a good reason for the administration of hydrochloric acid after the meals. As Dr. Sidney Martin has insisted, pepsin may be needed, hydrochloric acid is certainly needed; pepsin is a more persistent element in the secretion, and, moreover, as a ferment its work is not, like the work of hydrochloric acid, in direct proportion to the quantity present. Dr. Soltau Fenwick (34), Dr. Bardswell, and myself have pointed out that in phthisis, even of moderate severity, some dilatation is frequently present, especially in protracted febrile states of patients confined to bed, when in later stages the greater curvature may extend below the navel.

Dilatation, then, is variously associated with catarrh, as cause, concomitant, or consequence. When the cavity is not thoroughly emptied,

a remnant of decomposing food may taint all that comes in. But this retention is at its worst in cases of the class *B*, in which there is some *obstruction* at or about the pylorus. It is in these, whether of cancer of the pylorus, of fibrous thickening of the part, of external adhesions, of torsion, of cicatricial closure due to past ulceration or corrosive poison, that dilatation reaches its greatest degrees. It is in pyloric obstruction that the stomach attains those huge proportions which compel the patient to deliver himself of vomitings which fill a bucket. The stomach, instead of yielding, sometimes contracts, its coats becoming enormously thickened and its cavity reduced. In a patient of my own, aged about forty, in whom the symptoms pointed to a greatly thickened and contracted stomach, this state and pyloric stenosis were found after death. The stenosis was due to the scars of old ulcers. The walls were of extraordinary thickness and the cavity very small. There was no carcinomatous infiltration, the thickening was largely cirrhotic, yet during life peristalsis was very active and visible (*vide* p. 438).

By torsion we mean that a distended and overloaded stomach, dragging at its pyloric attachments, causes acute flexion there, whereby the issue of its contents in the normal direction is impeded. The late Sir W. Broadbent has added to this cause of increasing gastric dilatation the supposition that in some persons the lesser omentum is too short and the suspension of the pylorus unduly high; thus the kink at the pylorus would form more readily, and such persons would have a predisposition to dilatation of the stomach. But concerning dilatation of the stomach associated with visceroptosis the reader is referred to the articles on "Visceroptosis" (p. 860) and "Nephroptosis" (Vol. IV. Part I.).

Morbid Anatomy.—It is rather difficult to deal with this part of the subject without travelling out of it. The various causes of mechanical dilatation—whether cicatricial, fibrotic, or carcinomatous, or due to pressure, or adhesions of external origin, and so forth—are described elsewhere. In like manner, catarrh of the stomach and atrophy of the organ, although often associated with dilatation, are not an essential part of it, and, moreover, are described elsewhere.

Of the dislocations of the viscus it will suffice here to say that, so long as the pyloric attachments hold, the lesser curvature may take a more acute bend; but the stomach does not fall as a whole. When, on the other hand, the pylorus is dragged from its moorings the lesser curvature dips towards the oesophageal line obliquely, passing through the line of the navel; so that the stomach occupies the left hypochondrium. Or a "wallet stomach," in which fundus and antrum lose their distinction, may occupy the hypogastrium. When the pylorus is kinked the obstruction may be indefinitely increased (*vide* art. "Visceroptosis"). The state of the coats of the stomach depends upon the nature of the causes: if the dilatation be due rather to obstruction than to atony, the reluctant muscular coat will thicken, and with its increase there is usually an increase of the mucous coat also. If the hindrance be of slow growth, and the muscular coat undergoes a parallel reinforcement, the cavity, as

I have said, may not be enlarged, but may even be diminished; and the mucous coat, perhaps also thickened, may thus be thrown into exaggerated folds—the *état mamelonné*. Professor Hamilton says: "The mucous membrane is thrown into innumerable pyramidal elevations with obtuse apices, each about the size of a lentil seed—probably caused by spasmodic contraction of the muscularis mucosae, and of course it is often catarrhal." But in the vast majority of cases the cavity is greatly enlarged, and the hypertrophied muscular bundles lying between the connective-tissue septa—themselves also much increased—are very prominent. As in compensatory hypertrophy elsewhere, the thickening implies a sclerosis. This process, probably identical with the fibrous condensation which we see in the pylorus itself, is presumably irremediable. In chronic alcoholism it is well seen, beginning in the submucous tissue, wherein, as well as in the proper muscular coat, the fibrous elements slowly advance at the expense of the functional elements. For obvious reasons the hypertrophy is first and chiefly seen about the pylorus, the main motor of the stomach, and there likewise the fibrous change is conspicuous; even when by strain and pressure the muscularis of the fundus is atrophied, we may still find muscular hypertrophy about the pylorus. In some cases, as I have said, the chronic and diffused thickening is carcinomatous; in others, the leather-bottle stomach is by no means always malignant, as my own experience can testify; and in this opinion I am supported by the records of Prof. Leith (p. 438), and of other observers.

When we turn from these static cases of gastrectasis to the acuter or more dynamic changes, or to changes which, if not very acute, are rather adynamic than obstructive in origin, we shall not find muscular hypertrophy. All the coats are thin, even wasted, as the partial obliteration of the secretory glands will shew; and as the mucosa is often the seat of a catarrhal process the muscularis is the less able to retain its natural resiliency. Mucus is found more or less abundantly in the cavity, and serves as a source of decomposition. The contents of the stomach are often very acid, a reaction usually due to the organic acids—lactic and butyric. It is alleged that hydrochloric acid is often present, even to excess; certain authors say, indeed, that in some cases hyperchlorhydria may be the efficient cause of dilatation; but the grounds for this belief are insecure, and the presence of the organic acids gives a contrary presumption. Broadly speaking, if in mere dislocation free hydrochloric acid may increase a little, in dilatation it tends to diminish, and with chronic gastritis becomes less and less.

Symptoms.—In stating that "atonic dilatation" is very much more common than physicians have supposed, I am supported by the testimony of Prof. Saundby and Prof. Barrs; moreover, in not a few cases, more especially in those of atonic distension without obstruction, the patient does not complain of any characteristic symptoms; in the midst of his other troubles some dyspepsia, flatulence, or gastric discomfort passes unnoticed, or is accepted as part and parcel of the general malady.

Recently, I saw a gentleman, advanced in years, in whom, after a very tedious but not unsubstantial amendment from pneumonia, collapse had suddenly set in; for a few hours the collapse was combated with some promise of success, but death was not averted. This calamity being difficult to explain, a necropsy was made, when dilatation of the stomach was found. Either the dilatation, which may have existed some time, had suddenly increased, or it had fatally embarrassed a weak heart. There was no stenosis or gross disease of the stomach. The late Sir W. Broadbent described similar cases. The tongue is often clean, perhaps too clean, and indented; though with catarrh or carcinoma it is apt to be coated or glazed. A careful physical examination of the area of the stomach should then form a part of our regular clinical methods. The symptoms of chronic dilatation of the stomach are often of a general kind only, such as feebleness, anaemia, emaciation, chilly extremities, red nose and malar prominences, thirst, scanty urine often irregular in its constituents, heavy aching limbs, mental depression. If the paresis of the stomach sets in during the height of the illness, the patient falls lower and lower; he wastes more rapidly, and his strength wanes, or fails to increase, more than the apparent factors of the case quite account for. Now the physician who does not forget the liability of the stomach to fail in the fight, may interpret the signals of gastric distress. In such cases, if recovery is won, it is at heavy usury; the patient creeps out of his chamber wan, languid, and emaciated, and may regain his health but imperfectly even in a year or two. Not only is the general health slow in returning, but the stomach itself is almost the last part of the body to regain tone and functional vigour; the patient enters upon convalescence pined and reduced, and may have to face the world again as a chronic dyspeptic. For many a month physical examination will demonstrate how hard it is for a hollow organ to recover its natural elasticity. And in less fortunate event the dilatation of the stomach is but one more shaft from the quiver of Azrael; discovered or undiscovered, it is a part of the company of death. In obstructive cases, when well established, we usually find a sallow and hollow-eyed face, abdominal distress, sour breath, dryness of the skin and other tissues, chilliness even to ashiness or cyanosis, subnormal bodily temperature, periodical vomiting, and (more rarely) certain peculiar nervous symptoms of a spasmodic kind, advancing to slight or severe tetany, and due to peculiar causes.

Pain.—In flaccid extensions pain is usually insignificant. In obstructive dilatation it is not the ordinary pain of dyspepsia, or is that pain with a difference. It is not felt especially after food; it is not associated with eructations or acid risings; it is not periodic or even very variable.[1] It is not the acute pain of gastralgia or of some cases of

[1] A stomach which is capable of frequent upward discharges of its contents, whether solid or gaseous, is not seriously dilated. I omit all allusion in the text to those cases in which sulphuretted hydrogen is thus discharged: the symptom is a very noisome one. The gas is antagonistic to the lactic acid fermentation: it is often associated with hydrogen and marsh gas, and Boas says it is a result of dilatation (9). The symptom is of rare occurrence and difficult to explain; but, to judge by the one case I definitely remember, it is not

malignant disease, it is rather a sense of weight or oppression; yet, in large obstructive dilatation, it is wearing and intolerable. This pain is due to the burden within the stomach, and is quickly relieved—for a time—when by vomiting or siphonage the burden is removed.

Thirst in obstructive cases is not less distressing than pain. With a stomach full of slop, the patient is burning with thirst, for absorption from the stomach or bowels is almost annulled, and the liquids drunk by the thirsty sufferer lie in the sac stagnant until rejected; the thirst which follows even ordinary occasional vomiting is well known to every reader.

The urine for the most part is scanty. In obstruction it is usually deficient in chlorides; often it is alkaline and contains triple phosphates. Dr. Sidney Martin tells us that it may contain excess of ethereal sulphates from the putrefactive changes; though such changes (as he observes) are more usual in the intestines than in the stomach. Albumin is not an uncommon impurity of the urine in cases of gastrectasis of the extremer degrees; or at an earlier stage albumosuria (peptonuria) may occur, as in carcinoma or ulcer. Acetonuria is occasionally seen in cases of dilatation of the stomach, and may be due to defect in carbohydrate assimilation; for a further account of this symptom, however, the reader is referred to p. 469.

The Bowels.—In obstructive cases dryness of the skin and indeed of all the tissues, and constipation of a most obstinate kind, are conspicuous — the faecal masses being shrunken and hard. Conversely, these symptoms should lead to suspicion of dilatation or other atrophy of the stomach. Diarrhoea, as we have seen, occurs in some cases of gastrectasis. I am again indebted to the late Sir W. Broadbent for the account of a lady, once under his care for gastrectasis, in whom the stomach would empty itself from time to time with a rush along the bowels. The recumbent posture seemed to be the means of enabling the viscus thus to unload itself; after retiring to rest the patient would have a sense of the gushing of fluid within her, and on seeking the closet a profuse liquid discharge would issue from the rectum. After such a discharge the stomach was no longer perceptible by the physical signs which had previously made it only too manifest. Ultimately an autopsy was obtained and non-malignant thickening of the pylorus without stenosis was found. In the upright position the pylorus was probably closed by acute flexion of the part at the point of suspension. In atonic distension of the stomach again a lienteric discharge from the bowels is not uncommon, but may be a result of chronic gastro-enteritis.

characteristic of dilatation. It may be due to abnormal patency of the pylorus, so that intestinal gases rise into the stomach. The case I refer to, I saw with the late Mr. Copley of Wisbech in a neurotic young woman. The stomach was not permanently dilated, and Mr. Copley, by test lavage, believed it occurred independently of albuminous or any special kind of food. Indeed, the phenomenon would recur after the stomach had just been washed out and was presumably empty. Indol has been detected in the stomach in rare cases, and sulphuretted hydrogen, like indol, may be due to the presence of the colon bacillus (70). Hoppe-Seyler analysed the gases in eleven cases of gastrectasis, and found varying proportions of hydrogen, nitrogen, carbonic acid, and oxygen. The gas was usually inflammable.

Emaciation.—In almost all cases, whether of atony or of obstruction, there is loss of flesh. But the emaciation is not so regularly progressive as in malignant disease: and although even in malignant disease some temporary restorations of nutrition may be seen, yet in atony and the benigner forms of obstruction the condition is far more amenable to treatment. The late Dr. Sutton tells a horrid story of an autopsy on a case of his own in which he found food impacted layer upon layer in the stomach and gradually so consolidated there that the cavity was represented only by a small passage, in the middle of the mass, through which fluids had trickled scantily into the duodenum. This may serve as an extreme instance of the cessation of all nutritive function in the organ; and if in the dilatation of obstruction the state of the ingesta is usually rather that of a sour "swill," the absorption of fluids is none the less in abeyance, and none the less is the patient's body reduced by a dry atrophy. Moreover, such matters as may be absorbed are by the abnormal decomposition reduced to almost worthless elements. The appetite is bad or capricious. In simple atonic distensions these symptoms are present in some measure, but far less urgently. Moreover, the emaciation is often due as much to the primary conditions as to the state of the stomach itself.

Temperature.—As in other cases of reduced vitality, the temperature often ranges below the normal. For lack of fuel the warmth of the body is spent and not replenished. The patient feels chilly, is cold to the touch, pinched in face, and even ashy or cyanotic. The pulse is often slow, it is always feeble. Such changes may, on the other hand, be due in part to auto-intoxication. In children, however, acute tympanitic distension with epigastric tenderness is often associated with periods of febrile temperature and sharp transient attacks of gastric catarrh.

Vomiting is not a constant symptom, even in obstructive cases, unless the obstruction be tight and persistent. The small part taken by the stomach itself in the act of vomiting becomes probably less and less as the stomach dilates; the reflexes arising from its interior surface are weakened, and until the irritation becomes extreme the vomiting machinery is not called into action. Speaking generally, vomiting is periodic and characteristic in cases in which dilatation has come on gradually from mechanical causes, in the midst of comparative health; though in atonic cases in which the viscus has yielded under the shadow of exhausting constitutional maladies, it is far from unknown. But in atonic cases the vomiting is not periodical but occasional; it is not pumping nor voluminous, but consists rather of mucous froth and a little fermented food, as if an intercurrent gastritis were the immediate cause. In a lady now under my care for persistent gastric distension in neurasthenia, the consequence of domestic sorrows, such vomiting is no infrequent occurrence. In obstruction the vomiting is not frequent, it does not return after most of the meals, as in cancer without obstruction, but the meals even of three or four days accumulate. Moreover, individuals differ a good deal in readiness to vomit. Again in obstructive

cases eructations of food are unusual; the stomach, now unable to expel small quantities from time to time, when its burden becomes intolerable, is passively emptied by a sudden impulse of the abdominal machinery. The dyspeptic who suffers from acid or offensive eructations probably has no static dilatation of his stomach.

Whatever the history of the disease, vomiting in obstruction is a conservative process; by thus unloading himself the patient gets along. Under the weary education of affliction the abdominal muscles become trained to the new labour, and at a certain height or at a certain weight of gastric contents vomiting promptly begins. True nausea is not usual in obstruction; the contents of the sac are cast up by contractions of the abdominal muscles so violent as to shoot the foul stream from the mouth as from a pump. As if by repeated strokes of a piston, the spouting fills the pail until the onlooker wonders whence it all can come! The vomit, if held up to transmitted light, is usually darkish-grey in colour, and streaks of a lighter grey mucus are suspended in it: on the top is a darker, brownish froth: at the bottom is a deposit of solid matters; chiefly food-remnants mixed with mucus, sarcinae, yeast cells, and other microbes. By the microscope, or with the naked eye, matters swallowed many days or even weeks previously may be found. In the washings of the stomach of a woman suffering severely from gastrectasis, the late Sir W. Broadbent found (private report to myself), on a certain 31st of March, a remnant of preserved ginger which had been eaten on the previous Christmas Day. She was certain that she had never eaten of ginger since this day. In some cases hydrochloric acid is to be detected, often indeed in no small quantity (Cahn and v. Mering): it seems, therefore, that in these large accumulations hydrochloric acid cannot always arrest fermentation. The vomit probably contains also, as we shall see, certain poisonous products of which little is accurately known, but which may be charged with peril to the patient nevertheless. Unfortunately the vomiting, violent as it may be, does not quite empty the cavity; and, as in the case of other hollow organs, a little puddle remains to contaminate all food subsequently taken in. The relief, however, such as it is, is welcome: for some hours, or for a day or two, the sufferer is freed from urgent distress. In the cases described by Kussmaul, under the name of "Tiefstand," when the stomach, by means of tight lacing, or other dislocating agency, is thrown into a loop so that the pylorus approaches the cardia, the smaller curvature becoming acute, the large curvature extending, and the whole organ falling deeply down into the abdomen (*ride* art. "Visceroptosis"), vomiting may become difficult or impossible; so that siphonage is of urgent importance. Yet even in sling stomachs descended to the hypogastrium, vomiting takes place, in most cases, vigorously enough.

Nervous Symptoms.—Vertigo, which is a characteristic symptom in atonic distension, may not be toxic; often it seems due rather to some reflex process. Trousseau in his emphasis on *vertigo a stomacho laeso* was probably observing cases of atonic distension. It is usually relieved

almost at once by the dry, non-fermenting diet recommended on p. 548. But besides the depression of spirits which cannot fail to be present in an abdominal malady so persistent and so full of misery, there are, in not a few of these patients, symptoms which suggest a more specific cause. The weary and disheartened sufferer falls into a melancholy; he is annoyed by strange sensations; he is fretful, sleepless, and subject to nightmares; his head aches, and with the headache may be tinnitus, vertigo, or visual disturbances: he then may fall into a syncope, or, worse than this, tetany or convulsions may appear, which if complete or leading to coma are nearly always fatal (Trevelyan, Robson) (*vide* art. "Tetany," Vol. VIII. p. 47, 1899). Trousseau's observations of tetany in children are well known. These more alarming and indeed perilous conditions, although they are peculiar to children or persons whose nervous controls are reduced by starvation, seem to depend upon the formation of poisons in the stomach; Ewald, indeed, in a case of tetany of intestinal origin, discovered a kind of ptomaine in the urine. Senator was perhaps the first to suggest the possibility of this kind of auto-intoxication. M'Kendrick and Halliburton and Bouveret and Dévic injected fluid from the stomachs of such cases into animals, and the latter observers set up tetanic convulsions thereby. They state that, even in cases of such severity, an excess of hydrochloric acid will altogether prevent the formation of these poisons. Bouchard's arguments on auto-intoxication we cannot ignore, even if we think some of them exaggerated. Other physicians also have insisted upon some such explanation of phenomena, which in their origin are as obscure as they are lethal. Hitherto, however, it must be admitted that experimental and chemical researches on the subject have led only to negative and contradictory results, for Müller sought for toxic bodies in the dilated stomach of such a case with negative results; and although Brieger's peptotoxin can be obtained in the laboratory from fibrin, it has not been found in the living stomach. Lorenz, von Jaksch, and von Noorden have found acetone in the stomachs of persons apparently poisoned from their own organs; acetone might arise out of milk fermentation, but as on absorption it is more or less oxidised it could scarcely accumulate in dangerous quantities. Nor can any great harm come of the decomposition of fat or of carbohydrates; to generate toxins we look rather to albuminous ingesta, but such a conversion must surely be checked in the presence of a large quantity of acid, even if the acid be organic; and, as a matter of fact, such products of albuminous decomposition as phenol and indol are very rarely found. Alcohol is said to favour the occurrence of tetany; and till this observation is refuted it will be our duty to avoid alcohol, if possible, in our dietary or treatment. Alcohol, again, may be mischievous indirectly by facilitating absorption; or, as one of the few things which are absorbed rapidly from the stomach, while passing into the blood it might promote a contrary osmosis into the cavity. In discussing the causes of a poisoning which at least is to be recognised in its effects, we are reminded forcibly of Bunge's view of the stomach,

not as the chief organ of nutrition, a function which he attributes rather to the intestine, but as the seat of the protective disinfection of ingesta before they go farther. How important the stomach chemistry is in the prevention of such dangerous poisons as the typhoid and the choleraic is well known. It may be, therefore, that the brew of poison is due negatively to gastric inefficiency rather than to positive concoction. In the Czerny-Kaiser experiments, when the stomach was removed from dogs, the animals were poisoned by foul flesh which in the normal state they would have devoured with impunity; and Pawlow observed a like result in dogs after section of the vagi. Some recent experiments, however, have thrown doubt also upon this gastric factor in disinfection; moreover, tetany may arise in dilatation of the colon, and vanish or relapse as the colon is washed out or left to itself (Langmead and Garrod).

The notion that tetany is due to the mere desiccation of the tissues is unsupported. Bamberger, although believing that auto-intoxication is at the bottom of tetanoid accidents, while reviewing among other possible causes a reflex irritation arising from the walls of the stomach, dwells upon the desiccation of the tissues. The most important "desiccation," surely, must be the thickening of the blood from imperfect absorption, which I have compared to that in cholera; and, as in this disease, it is apt to produce morbid phenomena of its own, possibly even tetany; certainly cramp of the limbs is common in gastrectasis. The prevalent working hypothesis, however, is that the cause consists in one or more soluble poisons, formed, or at any rate present unneutralised, in the stomach.

Tetany, or other acute nervous disturbance such as convulsions or coma, and death have occurred not a few times during lavage; and, beneficent as lavage is on the whole, it is alleged that outbreaks of tetany may be determined by this operation. Dr. Soltau Fenwick has carefully considered this practical question. That lavage creates the dangerous factor seems improbable, but it may determine the absorption of it; as Mr. Mayo Robson has observed, tetany begins with painful gastric contractions, which the mere contact of the tube may set agoing. But Dreschfeld and others have seen tetany appear spontaneously in cases of dilated stomach in which lavage had not been practised (*vide* art. "Ulcer of Stomach," p. 470). Whatever the mode of the generation or of absorption of toxins, if such there be, careful lavage should rather cleanse, and prevent evil, than be itself the cause of the tetany or convulsions, though it is conceivable that the washing or a mere intubation at some critical moment may start the process. It is the duty of the physician, therefore, vigilantly to watch for conical spasm of the fingers, or inward thumb, and from time to time to test the forearm reflexes. Happily these dangerous accidents are very rare. For fifteen years my experience of gastric lavage was very large, yet, except in minor degrees and in children, I never saw tetany or convulsions in such cases, either during siphonage or otherwise. Tetany has never—to my knowledge—been recorded in mere atonic distensions of the stomach.

Peripheral neuritis has been recorded as a consequence of dilatation of the stomach (Carr, Clemensha), in cases in which other causes, such as alcohol or specific infections, were excluded. In Clemensha's case there was also an optic neuritis.

Finally, Bouchard describes an arthritis in patients suffering from dilatation of the stomach, and attributes this also to the absorption of poison from the alimentary canal: I cannot call to mind any instance of this sequence of events.

Physical Signs.—*Inspection.*—Emaciation is so commonly associated with gastrectasis that inspection of the abdomen alone often tells the tale. Not infrequently the outline of the distended organ can be seen, and demonstrated to a class; the breach thus made in the symmetry of the contours of the abdomen is very characteristic, the fulness usually lying towards the left hypochondrium. The abdomen need not be protuberant as a whole, and usually is not. The light should be at the head or foot of the bed. Until in the upright position "visceroptosis" has to be considered, the patient must be recumbent, with a pillow placed under his knees to slacken the abdominal muscles: for in the upright position the resonant area is usually diminished. With a high diaphragm the normal stomach may reach the fifth rib (Stacey Wilson), but in this case the resonant area is constant for all positions. Nevertheless this cause of a rise of the stomach upwards must be considered in the light of all the conditions of the case. When the main contours of the stomach are visible, and especially the upper contour, the organ is deranged, yet it may not be dilated; it may be simply displaced, or contracted and thickened. The lower curvature should cross the epigastrium about the line of the ninth costal cartilage, but if it does not transgress the line joining the iliac crests the distension may be within the bounds of the normal, or at any rate the stomach may be able to recover itself in due course; but in such cases the upper contour will usually be invisible. In many thin persons after a meal a too flatulent stomach may be seen and felt as a protuberance in the epigastrium; but in due time it recedes within its proper limits. Sometimes the diagnosis is facilitated by making the patient stand up and so placing him that the light falls obliquely upon the abdomen. Any variations between the vertical and horizontal postures will of course be noted.

If the larger curvature sweep down below the transverse line from 10th to 10th rib, rising again in the left hypochondrium up to the 10th rib or higher, and the lesser curvature, or rather the upper contour, be visible, there is more than a transient distension; the stomach is probably stretched beyond its capacity of resilience. In gross feeders the abdominal walls are usually too fat to permit the outlines even of a big stomach to be seen. In cases of dislocation, usually of course with more or less extension and dilatation, the lower margin may sweep down to the pubes: the liver and kidney may be displaced and palpable, and the colon also pulled downwards. If the stomach be thickened it can be grasped easily enough, or adhesions of the pyloric portion to neighbouring

structures detected, or condensed tissues around an inveterate ulcer palpated, as in a case I recently saw with Mr. Mayo Robson. Here again, however, the possibility of a mere displacement without great dilatation must not be forgotten. The position of the upper contour and the other facts of the case will probably prevent error in this respect. The gastric resonance crosses the median line—save for an inch or so—in very few cases, normal or morbid; in displacement the prominence of the cushion-like stomach below its normal position usually contrasts plainly enough with the "scaphoid" epigastrium, left hollow by its descent. The upper border often travels up and down with the breathing, or seems to do so. In the hollow the abnormal aorta beats very palpably, and many cases of "throbbing aorta" in neurotic women are of this kind.

If the outline of the stomach be not visible, or be indistinct, or its volume not sufficiently manifest for diagnosis, further means must be employed. The stomach may be revealed by administering to the patient an alkaline carbonate followed by an acid, so that gas may be given off and the stomach inflated. Most physicians, like von Ziemssen, prefer this plan to any other; he gives, in the more largely dilated stomachs in adults, about 100 grains of sodium bicarbonate and 80 of tartaric acid; but in moderate cases even a drachm of bicarbonate of soda, followed by half a drachm of tartaric acid, in small quantities of water, will suffice to throw the sac into relief. With care this method is neither injurious nor very uncomfortable, as Prof. Saundby testifies, who also prefers this method to inflation by the air-ball. To the emptied or fasting stomach he sometimes administers even as much as two drachms of bicarbonate of soda and a drachm and a half of tartaric acid. Other authors protest that insufflation by the air-ball and siphon tube is far better, as by it the quantity of air can be readily increased or diminished; but in the ordinary course of private practice these advantages, whether for inflation or for chemical testing, do not compensate us for the inconveniences of intubation, which must be preceded by lavage. However essential the tube may be in therapeutics, by careful palpations and percussions and by clinical observation, diagnosis may be adequately made without it. In atonic dilatation with slack pylorus these inflation tests may be defeated by the rapid escape of air into the bowels. Pepper and Stengel think that by inflating quickly at first a spasm of the pylorus is set up which prevents this escape. At best the distension is fugitive, and the observations must be made as quickly as possible.

To try to map out the stomach by pouring a large quantity of some fluid into it is to forget that the stomach is movable as well as distensible, and often very tolerant; but much assistance may be obtained in palpation for splash by administering a glass of water just before examination. Manometers have been contrived for the estimation of the elastic pressure of the walls of the viscus, but they are idle toys.

Peristaltic movements of the stomach are often to be seen in obstructive cases, especially in cicatricial pylorus. They must be watched for

before and after food: and if movements do not occur during inspection they may often be elicited. Flicking with the wet end of a towel is said to be a good way of producing them; but if the stomach be capable of them, manipulation usually suffices. A tumour the size of an orange may rise in the cardiac portion and travel slowly and sinuously towards the pylorus; a movement which may be repeated either spontaneously or under the handling. Reverse peristalsis is occasionally seen, and is significant of pyloric obstruction, but in the main peristalsis from left to right will prove to be gastric, and peristalsis in the contrary direction colic. In most cases, however, the sinuous movements of the walls are not continuously visible as such; but from a flat epigastrium a large boss or phantom tumour may suddenly form under the eye, and falling again reappear at another part of the gastric area, the intermediate progression, if such there be, being lost to sight. A soft and flat epigastrium may thus at intervals "rise up like a quartern loaf."

Palpation.—The hand passed very lightly over the abdomen may detect a prominence of the stomach when the eye fails to do so; for in these cases the abdomen is usually slack. In the larger number the nature of the prominence is scarcely to be mistaken; the organ gives to the hand a sensation as of an air-cushion, which is very characteristic. If peristalsis arise under the hand at the same time the diagnosis is easy; but the gastric walls may be too slack, or the contents not such as to produce this action. Sometimes, however, hand-pressure upon any tumidity about the pylorus will force up the sac into a boss. Movements of this kind become apparent only in cases of a chronic course, in which the coats of the stomach are hypertrophied; in cases, that is, of pyloric obstruction of whatever kind, whether the stomach be dilated or contracted: on the other hand, it is probable that palsy of the gastric walls is never complete except in very extreme conditions, such as collapse. The pylorus is, in my experience, never palpable except under morbid conditions; even if it be granted that the abnormality be only spasmodic, the condition is more than one of transient constriction; for instance, the stomach may have swung from its duodenal connexions to the left ("vertical" or "subvertical" stomach); but normally the pylorus has not more than a 2 cm. shift. A healthy but dislocated pylorus lying, let us say, across the line of the right rectus above the navel—may, however, be perceptible enough, and contract and relax under the hand, perhaps with borborygmi, in periods of some 30 seconds or so, thus simulating the large pylorus of infancy. In a case I saw in March 1907, the pylorus was to be felt in all its phases of contraction and relaxation quite vertically—that is, to the left of the left rectus and below the iliac line. On operation were found two chronic ulcers, but a normal pyloric orifice. A stable transverse body palpable in the "scaphoid" space above a dropped stomach is probably the pancreas, or at any rate must not be mistaken for the pylorus.

The "splash" is sought for by placing the right hand under the patient's left flank, and the left hand about the navel; the two hands are

then sharply thrust to and fro, either alternately or together; or the two hands may be placed on the abdomen, and the sudden displacements repeated; or again by grasping the iliac crests the abdomen may be shaken as a whole. A little patience may be required as, even in a dilated stomach, a definite splash is not often obtained at first; it would seem that some peculiar disturbance of the gaseous and fluid contents is necessary to produce it: the splash once obtained, however, may usually be reproduced at will for some little time. I am disposed to say, in spite of contrary opinions, that if a definite splash or slop is obtained, some substantial extension of the stomach, or colon, exists. The difficulty is that squelchy sounds may be produced which may indicate flaccid distended walls, but are not conclusive as to dilatation. Practice alone will enable the physician to distinguish between the mimic squelches and the unmistakable splash. The former are not uncommon in women, or in spare, dyspeptic, long-chested, nervous persons of either sex, who are also subject to the so-called crying bowels—spontaneous and long-continued gurglings which are sometimes so annoying as to drive the sufferers out of society. Many years ago I pointed out that in some neurotic girls some such sounds are rhythmical with the respiration, or are called forth by deep inspiration. In them the respiration, even when forced, is almost wholly diaphragmatic, and the troublesome disorder may be dispelled by promoting the expansion of the chest. In patients such as these manipulation often brings out squelchy sounds which simulate the splashing of gastrectasis, but they are not identical with its unmistakable variety; the wave is not dashed to and fro against the hands. Symons Eccles advised that the stethoscope be placed on the upper limit of the gastric resonance, and the flat hand laid upon the hypochondrium and sharply pressed inwards and upwards, when the wave of fluid, if present, will be audible, to be lost instantly on shifting the instrument just outside the line. On the other hand, I do not admit that even these equivocal, washy, squelchy sounds are to be obtained from the healthy stomach, even in thin persons, or after heavy flatulent meals. If the stomach be adapted to its contents, splashing or gurgling should not occur. I have tried scores of times to obtain them in myself, and I have failed to elicit them in any patient whom I could wholly acquit of gastric atony. Still, between such sounds and those which may indicate moderate degrees of distension, it is true that doubt may arise, or distinction be impossible; for instance, in chlorotic girls, in whom degrees of gastric flaccidity are not uncommon. A splash may often be produced in the colon, or possibly in the small intestine; in such doubtful cases the splash should be sought before food is taken, as for instance in the early morning, after the patient has drunk a glass or two of water. Flaccidity of the muscular wall of the abdomen, of course, facilitates the examination; but a "board-like abdomen" rarely conceals a dilated stomach. When the stomach is pressing up the diaphragm and encroaching upon the region of the left thorax, the heart-sounds often take on a consonating or metallic quality,

but this clang may not be indicative of more than a temporary windiness.

Percussion.—This means is more valuable in detecting gastrectasis than is generally admitted; the difficulty, especially in the obscurer cases, often lies in the distinction by percussion alone between resonances of stomach, of colon, or of distended small intestine. The colon, indeed, may override the stomach. By percussion after inflation the upper contours especially may be defined, and from this some estimate may be made of retained ingesta and secretions: in a few cases a shifting line of dulness, as of shifting contents, can be detected when the patient rolls over, or changes from the standing to the supine position. Usually in the upright position the stomach recedes from the surface and the percussion area is much reduced. In no case, of course, does percussion reveal the border of the smaller curvature, though in dropped stomach this portion of it becomes more and more apparent. The lower line of resonance should not descend lower than two fingers' breadth above the navel at any hour, and the emergence of the antral portion from below the liver is always morbid. Prof. Osler states, on Pacanowski's authority, that the greatest vertical diameter of gastric resonance in the normal stomach varies from 10 to 14 cm. in men, and is about 10 cm. in women; but to measure the capacity of stomachs in general by so many centimetres of resonant area on the surface of the abdomen is very fallacious. There is no "stomach note," properly so called: with the tone and tension of its walls the resonance varies through a wide compass, though in most cases in which stomach and colon are both inflated a difference of note or clang on travelling from the one surface to the other is notable enough, especially if one of the notes is carried into the right hypochondrium. In some such cases the area of hepatic dulness is diminished by this encroachment of the hollow organs. In atonic cases the area of gastric resonance, instead of descending, usually lies rather above than below the normal position. A common feature is the rise of the vault of the fundus beyond the normal line of the 7th (or at highest the 6th) rib, to the 4th, and laterally to the posterior axillary line. In dropped stomach of course this resonance is removed. The resonance rarely transgresses the middle line, or but for an inch or two, though in some cases the antrum so expands as to be confused with the fundus in one uniform sac. At no time after food does a flaccid stomach grip its contents so as to afford no resonance, but it is most extended some little time after a meal.

Auscultation alone is of little or no use. Combined auscultation and percussion affords a method of mapping out the stomach or colon which by some observers is highly valued. I have not succeeded in making much of it, nor of coin-tapping; but the fault may well be my own. It is practised by stroking, scratching, or tapping the abdominal wall radially outwards from the bell of the stethoscope placed over the area presumably gastric. (For the signs obtained by siphonage *vide* subsection Diagnosis, below.)

Diagnosis.—From what has gone before it is clear that the diagnosis

of dilatation of the stomach must be made by looking at the abdomen, by handling it, by puffing out the organ with carbonic acid or an inflator, and by percussion with and without simultaneous auscultation. The physician will also have the help of the symptoms and history of the case. By these simpler means in the large majority of cases he will be able to form a fairly correct opinion. But in baffling cases, especially in those in which surgical operation is proposed, to these aids we may add a skiagraphic picture, and an investigation of the contents of the organ by siphonage—perhaps the most valuable, if least acceptable, means in obscure cases. First of all, in passing the tube the position of the cardia may be ascertained; it is said variously to lie at spots between the 9th and 12th vertebrae; no doubt its site is variable. It rises and falls 3 cm. with respiration, and is apt to be dragged down if the stomach is weighted with much fluid (M'Caskey). Time after time to find considerable remnants of food in the viscus seven or eight hours after a meal gives a strong presumption of motor inadequacy. A moderate meal is dispersed by that time, even by a slack stomach; a light dinner should be disposed of long before the lapse of twelve hours. A few cubic centimetres (20 to 30) may be removed in the morning, but this residue should never contain relics of food, scarcely even in microscopic quantities. In a mild case the stomach is not empty in five or six hours, and in severer cases, especially when feeding has been too solicitous, even after twelve hours arrears are not wholly disposed of. Luncheon encounters the relics of breakfast, and tea and dinner are piled upon the relics of both. Yet I would not pretend that the presence of remnants even after twelve hours, however significant, is necessarily of serious import, however helpful as a guide to treatment.

As regards the further analysis of the contents of the stomach, as by the method of test meals and so forth, I regret to say that the hopes, and even the confidence, of our chemical brethren have fallen something short of full justification. In too many cases chemical reports, occasional as they generally are, prove fallacious. Correct, no doubt, for the phase and hour of examination, such a report, or even a group of occasional reports, is to be valued as no more than one of many signs and symptoms to be considered comprehensively and together, and, unless based upon more frequent, various, and systematic series of tests than is ordinarily practicable, must not be allowed to prevail over trained clinical judgment. In a certain elderly patient, examined by three or four distinguished physicians, occasional reports on test meals declared a lack of free mineral acid, and malignant disease was apprehended. The case proved, on the contrary, to be one of hyperchlorhydria, and a fair recovery was made. The chemistry of the stomach is subordinate to biological variables (*vide* p. 401, "Neuroses of the Stomach").

A progressive diminution in the average quantity of urine, and desiccation of the tissues, suggest a tight pylorus. If bile be found in the stomach the pylorus is more or less permeable. In obstructive cases peristalsis is likely to become visible. In gastroptosis the lower border of

the viscus is or may be manifest; the upper border is less definite. An upward lift of the stomach by a high diaphragm or by retraction of the left lung would be recognised by the other features and the history of the case. A fallen stomach is not necessarily a dilated one, though by kinking in the pyloric region it is in great danger of becoming so. In ptosis other viscera, especially the kidney and colon, will probably be displaced also. Illumination of the stomach from within, as practised by Einhorn, and by Reichmann and Herying (gastrodiaphany), has not fulfilled its promise; the rays undergo so much diffusion as to render the method fallacious. If substantial tumours occupy the stomach some opacity to the rays might make them manifest, but rarely could lead to their discovery.

The absorption methods again are of little practical value. If to a patient with dilated stomach potassium iodide be administered, the drug, instead of appearing in the urine in some ten minutes, may take an hour and more to present itself. Chlorides, again, abundant enough in the stomach, would be scanty in the urine. In using such tests as these, however, it must be remembered that rates of absorption and elimination depend on too many variables to be uniform, and where the delay is considerable the diagnosis will have been made by more direct means.

Of the value of radioscopy in definition of the size and place of the stomach I have little or no personal knowledge. Prodigious doses of subnitrate of bismuth (six to thirty ounces!) have been given by mouth or tube, it is said with impunity, and its shadow noted accordingly. The observation should be made quickly and the bismuth then siphoned out. Intra-colic injections have been made in like manner and the areas compared. More manageable means, it would seem, are the introduction of a large and insoluble bismuth pill, which, as it is said, will roll about in the stomach as the patient is turned from side to side, so that the curve of its successive positions may be recorded on the skin. Others (Dalton and Reid) recommend the insertion and manipulation of the ordinary tube with a solid metal end, or charged in its lower length with bismuth, the skiagrams of its successive positions being collated.

If the stomach be very capacious downwards, its distension is due to mechanical causes; if oedema of the feet or shins be present, free hydrochloric acid absent or very scanty, and lactic acid present without milk or bread diet, the mechanical cause may be cancerous, though not necessarily of the stomach itself, and occult blood may be sought in the stools. On the other hand, an atonic stomach, virtually dilated, often, when almost empty, recovers something like its normal limits, when effervescing powder or siphonage will settle the question. A stomach which, whether large or not, balloons on a moderate dose of effervescents is atonic. The diagnosis of malignant obstruction, if no tumour be palpable, must depend on the age of the patient, on his clinical history, and only in some degree on estimations of hydrochloric acid.

For some remarks on the diagnosis of hour-glass stomach due to the scar of ulcer the reader is referred to p. 472.

The following 3 cases are good examples of the occasional difficulty of diagnosis:—Hayem reports the case of a man, aged 42, who was admitted suffering from dyspnoea, cyanosis, cold extremities, and oedema of the legs. A rasping murmur was heard in the 4th intercostal space, and at the apex a humming bruit like a mill-wheel, not synchronous with the heart sounds. The urine was very scanty, but contained no albumin. The abdomen was distended, very painful, and tight, as if peritonitis were present. The stomach was now found dilated to a line some inches below the navel. Lavage was used daily with great relief; the urine was trebled in amount, and the cardiac murmurs ceased. After death stenosis of a non-malignant kind was discovered at the pylorus; in this case it would seem that the stomach arrested the diaphragmatic movement, pushed the heart upwards, and even deformed its chambers.

The second case I saw with Dr. Laurence Humphry. A middle-aged lady fainted, fell into collapse, and died within twenty-four hours. A thorough examination of the patient was impossible, but we detected, as we thought, considerable dilatation of the stomach. A good tossing splash was readily obtained, there was a tympanitic area and a definite cushion-like tumidity. The acute dilatation of Fagge was discussed, but the symptoms did not point to this alternative. On post-mortem examination the bag proved to be an enormously dilated transverse colon; the stomach was small and retracted, and lay high up under the ribs.

In old or cachectic persons the stomach may be atrophied in both mucous and muscular coats, distended and even dropped, with few or no direct symptoms. Mr. X., aged 66, when seen by me (December 20, 1897), was emaciated and jaundiced. There were "cushion stomach," splash, and extension of gastric resonance to the 5th rib and posterior axillary line, and downwards, towards the transverse iliac line. On siphonage the fluid ascended with difficulty on epigastric compression. Carcinoma of the liver was diagnosed secondary to that of the pylorus, but at the necropsy though cancer was found in the biliary passages, in the stomach there was none.

Prognosis.—This must depend greatly upon the causes and the degree of the dilatation, and no less upon the date of the diagnosis. In my experience obstructive dilatation of the stomach is a very obstinate and too often an incurable malady, unless relief can be given by the surgeon. On the other hand, after perseverance with lavage, largely dilated stomachs not rarely diminish and brace up until, if not a cure, a very substantial amelioration is obtained. If there be stenosis of malignant origin the prognosis will fall under that of the primary disease. In atonic dilatation without stenosis the prognosis, as a rule, ought to be hopeful, but the physician has no easy task before him; in atonic dilatation after exhausting disease, if the degree of it amount to a troublesome complication, the cure of it will be long and difficult.

Treatment.—We may have to deal only with an overworked or irritated organ becoming insufficient: its secretions and its motor activity

fail; food is delayed in the cavity and the viscus dilates. If so, how is this chain of events to be prevented? In dilatation consequent on stenosis there may be no great loss of gastric juice, and the motor defect may be a relative one only. In these cases Kussmaul's lavage has enabled us to be of service to a class of great sufferers. After a few test meals the physician will form some estimate of the quantity and value of the gastric juice, and digestive capacity. The necessity of siphonage will also be determined by the amount of residue found in the cavity at different intervals. If bacterial fermentation be active, carbohydrates, such as starch, dextrin and sugars, must be given sparingly, though not of course excluded. If catarrh be present, the siphon will reveal it. The conditions being thus determined, the dietary can be arranged. The first rule, and one good for all cases, is that liquids by the mouth shall be restricted, at any rate during digestion. As much of the fluid swallowed with a meal flows away in gushes through the pylorus to be absorbed elsewhere, if the pylorus be narrow or impassable, the ingestion of liquids adds a useless burden to the stomach already oppressed; and, by diluting the gastric juice, still further enfeebles its digestive powers. The quantity of food to be taken at each meal must depend upon the weight of the patient and upon his state of health; it should be divided into six small meals a day taken at nearly equal intervals. Overfeeding in atonic extension is obviously harmful, and as Dr. Bardswell in chronic cases of pulmonary tuberculosis of long standing found atonic dilatation in 50 per cent, and in febrile cases still graver dilatations, the need of caution in urging too generous a diet is imperative. M'Callum's observations are almost identical with Bardswell's.

In *obstructive dilatation* the diet must not only be "dry," but it must be administered finely divided, in small quantities, and as economically as possible; that is, it must be concentrated, digestible, and nutritious. The prevalent use of milk in these cases is not without disadvantages: if, however, auto-intoxication be suspected, and milk in this respect is desirable, one of the best brands of condensed milk without sugar may be prescribed; otherwise tender meat, lightly cooked and teased from the fibre with a fork, is lighter, more digestible, and more sustaining. Meat essences as a relish, the dry meat powders and jellies, artificial "proteins," minces, purées of chicken or fish and other tender meats may be allowed. Fat is so important an element of diet, especially in emaciated patients, that in the diet some fats must be included. Eggs—raw, boiled, or in custards—are nutritious and of small bulk. Cream, again, or a little fresh butter, may be both pleasant and useful. In the cases of stenosis in which the gastric juices are not much in defect, carbohydrates may be taken in gradually increasing quantity without creating the flatulence which is so mischievous in increasing distension at the very time when the stomach should be contracting. Vegetables are better avoided unless in very smooth purées and small quantities; if fresh meat be taken they are not necessary, or a little lemon juice

may be taken as required. Bread should be toasted, for toast is thinner, its starch-granules are ruptured, and the lactic acid bacteria are killed; such starchy foods as arrowroot, sago, rice, and the like should be diminished, the coarser cereals such as oatmeal forbidden. Jellies and blancmanges are very suitable and pleasant. If at first it is better to limit the starchy food to rusks and plain biscuits, as the patient's state improves malt extract may be added to the diet with advantage, as it is readily soluble, nutritious, and laxative. Honey, in so far as it consists of laevulose, is said to be less windy than sugar, or pure laevulose or saccharin may be preferred. Liquids cannot of course be forbidden altogether; small quantities of good old spirit, such as whisky or cognac, may be taken in a little water, if desired. Effervescing drinks must, however, be rigorously prohibited. Tea and coffee must be laid aside, but a little cocoa made from the nibs may be permitted.

The fluid withheld from the stomach must be injected into the rectum. About half a pint of water, warmed to blood-heat, may be thus used three or four times a day; a little table salt, and, in case of need, a little brandy, may be added to the injection. After a few days the rectum will accustom itself to the charge; but at first the fluid must be injected very gradually, a napkin must be pressed to the seat for a few minutes, and the patient should lie on his left side until the sense of bearing down has ceased. In a day or two the urinary excretion will be augmented, it will contain chlorides more abundantly, and the tissues will become softer and plumper.

In first undertaking an acute case of *atonic dilatation*, it is well to put the patient to bed and to feed him chiefly by the rectum for a week or ten days, with the usual precautions. In atonic cases the icebag is said to prevent gaseous distension, and in hyperchlorhydria to relieve pain. It is recommended to be worn almost continuously for ten or fourteen days. My experience of it in cases of other kinds would be rather in favour of intermittent applications. Other physicians as strongly recommend hot applications to the abdomen. A successful case was lately reported in which the patient lived for some long time and gained ground on chewing meat which when fully masticated was spat out, but he was occupied all day long in chewing his cud. Prolonged mastication is requisite in all cases, not only for comminution and insalivation, but to prevent the swallowing of air with the food. For this reason, and to avoid sepsis, the teeth should be kept in perfect order. In the atonic cases in which the gastric juice is more or less deficient, malt extracts and peptonised food are very useful. To promote the flow of gastric juices sapid foods must be offered; thus, although a dish of soup may not be permissible, a teacupful of hot beef tea may be a good introduction to dinner; but it is better not to give very tasty things, even if they seem digestible, for the abnormal delay of food in the stomach makes flavours, transiently not unpleasant, distasteful by their persistence or recurrence. For this reason I do not order cod-liver oil, although it is a valuable remedy. The rectum seems to have some

considerable power of taking up fat, and fresh suet smoothly mixed with milk may be given as an enema instead. The patient must be weighed regularly, at the same hour of the day, so that the fitness of the diet may be controlled.

The mode of life, such as fresh air and gentle exercise, and freedom from worry and fatigue, will be prescribed for each case as circumstances may dictate; but in respect of dress the strictest warnings must be given against all kinds of belts or corsets, for corsets are accused not only of aggravating visceroptosis, but even of causing it. A medically fitted abdominal bandage, on the other hand, is often very helpful and comforting in visceroptosis or weak abdominal muscles.

Siphonage.—We have seen that the information which lavage gives to the physician is often no less than the relief it gives to the patient; not only so, but familiarity with the practice has led us to extend its use to the investigation of dyspepsias other than gastrectasis. Disagreeable and even revolting as it appeared at first, the remedy is now quietly submitted to; physicians and patients become less shy of strange methods, as horses get used to bicycles and motor-cars; yet when, on the first appearance of Kussmaul's paper, I begged a lady of atonic fibre, afflicted with a gastrectasis due largely to an abuse of aerated waters, to allow me to wash the stomach out, I begged in vain. I found no great difficulty in introducing the practice into the Leeds Infirmary, although hospital patients resented it at first; very soon, however, men and women of refinement took to the use, if not indeed to the abuse, of lavage as naturally as after a like period of shyness they did to morphia injections. The danger was lest "autolavage" should be practised indiscriminately or habitually. The sphere of its operations has, however, much diminished during the last few years, because its systematic use is to be deprecated in atonic distension, and permanent obstructions are now dealt with by surgical methods. In 1869 the stomach-pump was our clumsy means of lavage[1]; soon afterwards a siphon, such as is now used, was made for me by Messrs. Harvey and Reynolds of Leeds, and the same improvement soon suggested itself to other physicians. The stomach-siphon is too familiar now to need minute description: it consists of a tube of rubber, or still better of a thinner woven material like that of which flexible catheters are made, with two lateral openings at the distal end. These eyes must be smooth, not quite opposite, bevelled inwards, and large enough not to get blocked by solid matters; beyond them the end should be solid. To the other end a large glass funnel is affixed. A short piece of glass tubing in the upper length enables the operator to observe the movements of the fluids. These siphons are now sold by all the best surgical instrument makers. In irritable pharynx—as of drunkards—a cocaine spray or a previous dose of bromide will facilitate the acceptance of the tube. The soft tube which falls into a curve on reaching the stomach

[1] Dr. Affleck introduced lavage by the stomach-pump into the Edinburgh Infirmary in 1871.

is of little use in measuring the depth of the stomach from the teeth; this information is to be got by other means. My first siphoned patient, or one of my first, was admitted into the Leeds Infirmary with benignant pyloric stenosis and large dilatation. Him I relieved so greatly by lavage that, being a sharp and inquisitive person, he set up as a quack on the strength of his experience, and washed out all the queasy stomachs of the country-side. I believe he made a very good thing of it; so much so that, being a grateful as well as an ingenious person, he offered me a share of the booty. His irregular practice had the advantage of proving to the public that the process is less repulsive than at first sight it appears.

I soon gave up the use of disinfectants in lavage, even in foul cases. If the cavity be washed until the water returns clear, our purpose is sufficiently attained. The stomach never was and never will be aseptic; perhaps it is not desirable that it should be. On the other hand, the use of antiseptics may have its own dangers. But if thick mucus be present the water should be made alkaline. Dr. Mercier has sent me the notes of lavage in a young man suffering from acutely suicidal melancholia. He refused food, and his breath was fetid. The gastric discharge was so offensive that the odour "like that of a guano ship" penetrated all over the large house. Thorough washing with plain warm water sufficed to clean his stomach, and thereon promptly to relieve his mental symptoms.

The purpose of siphonage is not only to clear the stomach of decomposed food, but also to cleanse it from the slime of catarrh, and from other fluids which seem to accumulate in larger quantity than the food and drink quite account for. For this reason, if the siphonage be practised, as is customary, once only in twenty-four hours, I think it is best on the whole to do it in the morning before the digestive labours of the day are begun. It is a common practice to wash out at night, but a morning wash-out will demonstrate that a great deal of tainted stuff finds its way into the sac during the night. Moreover, the results of the morning siphonage are a useful measure of the results of treatment. On the other hand, some restless sleepers find that to cleanse the stomach at bedtime secures a better repose. Sometimes there is great difficulty in emptying the stomach on account of the solid or semi-solid matters which block the eyes of the instrument. This trouble is generally worst at first; it disappears after the diet has been carefully regulated, and the state of the cavity ameliorated. If the eyes get plugged, an elastic ball may be quickly adapted to the tube at the place of glass insertion, whereby a pumping action may be carefully added to the siphonage, and the plugs gently forced one way or the other.

It is scarcely necessary to say that if cancerous or simple ulcer exist, the tube should be used with the greatest care, or not at all.

Unpleasant as the process must be, such is the relief of the pain and other symptoms, both local and general, in obstructive dilatation, or even in inveterate atonic cases, that patients often carry out the method with unfailing regularity, even after the vigilance of the physician is relaxed or

his attendance has ceased. Generally speaking, one siphonage in twenty-four hours is sufficient; sometimes, indeed, twice or thrice a week may suffice; on the other hand, in some bad cases a second cleansing in the later day is desirable: the frequency of the operation will depend on the amount of fermentation. It is needless to say that, as a rule, four hours at least must elapse between a meal and the operation. In cases of phthisis pulmonalis with gastric distension, siphonage once or twice a day for two or three weeks only is of signal service. It is also of service in relieving the vomiting of intestinal obstruction.

As a rule the water used in siphonage must be lukewarm; but at the discretion of the physician a final dash of cold water may be given as a tonic to stomachs which retain some power of reaction. In atonic distension care must be taken not to stretch the stomach by throwing any weight of water into it, but the irrigation must be continued until the water comes away clear. Prof. Osler says that it is not necessary to remove all the water; it has always been my practice to remove as much as possible. Dr. Sidney Martin advises that after siphonage the funnel be removed and pressure made upon the epigastrium to expel the residual contents; while this is done the patient should lie on his left side. I daresay that this is good advice, at any rate in cases of pyloric stenosis when even clean water cannot get readily away into the duodenum.

In acute (Fagge's) dilatation—which is apt, by the way, to occur after operations under general anaesthesia, and to be mistaken for post-narcotic vomiting—the patient must be turned at once on the belly or into the knee-elbow position, and while he is thus prone the tube must be passed, and carried as deeply as possible inwards, and epigastric compression applied. If relief is obtained the patient must not be allowed to turn supine for many hours, and then very tentatively. Under this management a few cases have ended in recovery. Surgical interference has hitherto proved ineffectual if not disastrous. Mr. Robson thinks gastro-jejunostomy is the operation to be preferred. Food, of course, must be absolutely forbidden, but large normal saline enemas are to be administered (*vide* also p. 769).

In ordinary atonic distensions siphonage, if required on occasion for diagnosis, is not a necessary part of treatment except in some toxic crises with vomiting, or in a few old-standing cases of laboured digestion, when a few weeks' of it often gives a relief to the organ which enables it to pull itself together. If there be evidences of chronic gastritis generous doses of bismuth must also be prescribed. In cases of extreme peril when collapse or exhaustion is present or imminent, or again in the last stages of organic disease, the introduction of the tube is, of course, to be deprecated: the operation itself increases the distress, and the results of siphonage are altogether disappointing; the stomach does not respond. Post-operative distension of the stomach, however, which is too often overlooked, is usually due to septic causes. The stomach should be examined in all equivocal cases, as lavage is often a successful part of the treatment.

Massage is easier to obtain than it was, but it remains still the advantage of a few; yet in gastrectasis of the atonic kind it is a valuable addition to our resources. Unfortunately, many as are the pretenders to massage, few are they who by training and natural endowment practise it skilfully. In the gastric atony of neurasthenic and emaciated persons the Weir Mitchell treatment is as indispensable as it is efficacious. In some cases improvement sets in fairly well, but, some months after leaving off the remedy, the patient may relapse. An incidental advantage of the massage treatment is the efficient control over the patient and all his ways. In his treatise on massage Symons Eccles testified to the value of the means in atonic dilatation, especially in cases of alleged neurasthenia. Massage is said to be potent to restore the qualities of the gastric juice; and cases of ectasis are said thus to gain much, not only by the promotion of nutrition, but also by dispelling any toxic matters which would otherwise lie in the body. That local massage can reduce the gastric cavity by reflex influence is a doubtful proposition, still its indirect advantages are substantial; for instance, its influence upon the constipation is favourable. I have seen "rectal feeding and massage" proclaimed as a cure for atonic dilatation, but I must warn the reader that a patient on rectal feeding only can bear very little massage. Indeed, it is too often forgotten that the massage is as much exertion as most patients can bear, and that while under it they must be kept torpid in bed, and dissuaded even from much reading or amusement.

Electricity may be useful in atonic cases, but as yet I have seen no definite good from its use, whether internal or external, except that a slowly interrupted faradic current may succeed in strengthening the abdominal muscles and promoting the action of the bowels; other physicians seem to have been more fortunate. Of the high-frequency current and "vibrators" in these cases we heard more two or three years ago than we hear now; perhaps because few physicians have the means or the time to carry out such exacting methods. The truth is there is a large element of "suggestion" in these imposing rites, an element which unluckily wanes as the public becomes familiar with them. Hydropathic means, such as packing followed by cold sponging, if very carefully instituted, are of service in debilitated conditions of which atonic distension is a feature; but the ordinary routine of water cure may be injurious.

Drugs.—The use of drugs to stop the vomiting of obstructive dilatation, in so far as successful, would probably be mischievous. Vomiting, nature's means of relief, is rendered unnecessary by lavage. I am strongly opposed, therefore, to the administration of large doses of alkalis and alkaline earths to neutralise the acidity, and thus to reduce the value of such mineral acid as may be needed. If stenosis be not invincible, saline laxatives, such as Carlsbad salts or plain sulphate of soda, are of great service; but the remedy is to be used discreetly. A draught of 20 minims of hydrochloric acid (B.P.), with a teaspoonful of the glycerin of pepsin and 5 minims of the liquor strychninae, may

be given after each meal to a male adult. Taka-diastase (gr. 3-5) is said to be potent in the digestion of starch, and in this sense may be antacid. Papain sometimes seems efficacious in cases of defective secretion, but it is very difficult to obtain the drug in an active state. In severe cases, at any rate for a time, some of the food should be predigested; I think, however, that in mere atony this aid should be gradually withdrawn as the stomach is enabled to do a little of its own work. Of other drugs nitrate of silver in pills is one of the most useful. The late Sir W. Broadbent recommended creosote and its congeners. In static dilatation none of these drugs is of use unless the stomach be regularly washed out, and in any case absorption of remedies, if this be desired, is slow and uncertain.

The warmth of the body is to be economised; these very chilly patients had better keep their beds until some heat-generating power is recovered, and the action of the heart reinvigorated.

In cases of persistent dilatation which does not yield to lavage, diet, medicine, massage, and electricity, and depends on some increasing mechanical obstacle, operative interference is indicated; it may prolong life, even in cases of slow malignant disease. But let not the surgeon be too eager, or the physician too diffident; when the obstruction is benign in nature, and not very complete, or in the dilatation of ptosis, the stomach, when once its arrears have been wiped off and it can start fair in a restored body, often recovers to a remarkable degree. In ptosis recumbency during digestion is often immediately helpful. It is reasonably advised that the bed or couch should slope a little downwards towards the head. Dr. Ewart and Sir W. Bennett published a case in which a reef was taken in on a dilated stomach (gastroplication), at least with temporary success; and a few other successful cases of the kind have been recorded by the late Mitchell Banks and others. In the vast majority of cases needing such heroic methods permanent obstruction is present and gastro-enterostomy more appropriate. For the treatment of hour-glass stomach likewise, treatises on surgery must be consulted.

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T. C. A.

ULCER OF THE DUODENUM

By the late Prof. JULIUS DRESCHFELD, M.D., F.R.C.P.

AN ulcer of the same characters as the gastric ulcer is found in the duodenum. The symptoms vary somewhat with its exact position. It is of these ulcers only that we can treat here. Ulcerations of a tuberculous nature, ulcers in enteric fever, and cancerous ulcers, which are occasionally found in the duodenum, are not included in this article.

Ulcer of the duodenum is less common than gastric ulcer. Its absolute frequency as estimated by Sir C. Perry and Dr. Shaw from an analysis of 17,652 autopsies at Guy's Hospital between the years 1826 and 1892 is 0·4 per cent. The Fenwicks found an open ulcer of the duodenum in 0·26 per cent of 13,055 autopsies. The relative frequency of gastric and duodenal ulcer is variously estimated as 9 to 1 (Trier), 10 to 1 (the Fenwicks), 40 to 1 (Andral). Occasionally an ulcer of the duodenum and an ulcer of the stomach are found together. The Fenwicks estimated that about 1·7 per cent of their cases of gastric ulcer were accompanied by a similar lesion of the duodenum. While it would appear from the experience of Moynihan and of Mayo that about half the cases of duodenal ulcer are associated with gastric ulcer.

Etiology.—*Sex.*—According to most observers it occurs more frequently in men than in women. The ratio being 10 to 1 (Krauss), 5 to 1 (Mayo, Collin), 6 to 1 (Robson, Weir), 2·5 to 1 (Mayo and Graham), 4 to 1 (the Fenwicks), 3 to 1, or if burns are excluded 6 to 1 (Perry and Shaw). In Mr. Moynihan's 114 cases there were 76 males and 38 females. Boas explains the preponderance of duodenal ulcer in males by the greater incidence of alcoholism and therefore of acid gastritis in that sex; the acid contents of the stomach passing into the duodenum so act on the mucosa as to reduce its vitality.

Age.—Duodenal ulcer may occur at any age, even in infancy. Dr. T. D. Lister records a duodenal ulcer in a child three days old, probably due to embolism from umbilical thrombi, and Spiegelberg one in a child five days old. Chvostek, in 87 autopsies on children under 10 years of age, found ulcer of the duodenum in 5. Oppenheimer has reported 15 cases of melaena neonatorum. In 279 cases Collin found the age-incidence to be as follows:—

Under 10 years .	42 cases	Between 40 and 50 .	46 cases
Between 10 and 20 .	24 „	„ 50 and 60 .	41 „
„ 20 and 30 .	43 „	„ 60 and 80 .	28 „
„ 30 and 40 .	52 „	„ 80 and 90 .	3 „

The Fenwicks, distinguishing between acute and chronic duodenal ulcers, find that 68 per cent of the former prove fatal between 15 and 30

years of age, and 63 per cent of the latter between 30 and 50. Ulcers due to burns occur much more frequently in women and children.

Occupation and Habits.—On this subject we have no trustworthy information. Alcohol is regarded by some as an etiological factor, but this is very doubtful.

Associated Diseases.—Sir C. Perry and Dr. Shaw collected 25 cases of ulceration of the duodenum in association with tuberculosis; of these, 11 only appeared to be tuberculous. These were associated with extensive tuberculous ulceration of the rest of the alimentary tract, and the duodenal ulcers were small, thickened at the edges, and shallow; the other 14 cases had more of the character of gastric ulcers.

Ulceration of the duodenum has been observed in chronic Bright's disease. Barié and Delaunay report a case with very severe intestinal haemorrhage a few hours before death, and have collected 18 cases.

Among other diseases in which duodenal ulcers have been found may be mentioned pneumonia, heart disease, septicaemia, and enteric fever.

Burns.—Curling first drew attention to the occurrence of duodenal ulcers after extensive burns of the skin; and he was able to collect no less than 10 cases. Since then the subject has been investigated by several observers, especially in England, with the result that, though the occasional appearance of an ulcer after an extensive burn has now been well established, the occurrence is certainly rare. The Fenwicks, on the basis of the statistics of T. Holmes, Erichsen, and Perry and Shaw find that ulceration of the duodenum occurs in 6·2 per cent of all fatal burns; Collin attributes 38 of his 279 cases to the same cause. It is further established that these ulcers occur chiefly in young subjects, and more especially in extensive burns of the trunk; that the ulcer occurs in the second or inflammatory period of the burn from the seventh to the fourteenth day, occasionally as early as the second day; that before ulceration occurs there is marked congestion followed by haemorrhagic infiltration which affects other parts of the alimentary canal besides the duodenum. In several cases (one noted by myself in which death took place six days after extensive burns) the lesion did not go beyond a haemorrhagic infiltration; in other cases an ulcer forms and may lead to perforation or to profuse haemorrhage. The formation of the haemorrhagic infiltration is most rationally explained by a septic embolus, and the change into an ulcer by the action of the gastric juice; a view which receives some support from Mr. Lockwood's experience of 138 cases of burns treated with all possible antiseptic precautions in which only one case of duodenal ulcer occurred. Dr. W. Hunter succeeded in producing extensive ecchymoses and ulcers of the duodenum in dogs by injecting toluylenediamine, and suggested, as a probable explanation of the duodenal ulcer in burns, the excretion, with the bile, of a poison which causes the ecchymoses and ulceration. Experiments by Ponfick and others have shewn that in animals extensive scalds will cause haemolysis similar to that seen on injecting toluylenediamine. Considering, however, that after burns ecchymoses are found in other parts of the intestinal tract as well as in the

duodenum, Dr. Hunter's view can scarcely be regarded as well established. Moreover, Dr. Fenwick found in dogs that ligature of the common bile-duct did not prevent ulceration of the duodenum after toluylenediamine poisoning. Cooke, on the basis of Weinland's researches on the existence of antiferments in the cells of the mucosa, suggests that a duodenal ulcer may result from destruction of these protective bodies in the mucosa by toxic substances appearing in the blood in superficial burns, or by excessive acidity of the gastric contents entering the duodenum.

Pathological Anatomy.—*Situation.*—In most cases the ulcer is in the first part of the duodenum, and most frequently close to the pyloric ring; less frequently it is in the second, and more rarely still in the third part of the duodenum. Out of 149 cases collected by Perry and Shaw it was found 123 times in the first part; 16 in the second part; twice in the third part: in one of these last the ulcer was probably tuberculous; in the remaining 8 cases all three parts were affected. In Collin's 262 collected cases the ulcer occurred within two inches of the pylorus 242 times, 14 times in the second part, and 6 times in the third part. In Mr. Moynihan's 114 cases, 107 occurred in the first part and 7 in the second. The ulcer is located most frequently on the upper portion of the anterior wall. Oppenheimer cites 15 cases of perforation, 11 on the anterior wall, 3 on the posterior, and 1 on the superior. In Collin's 118 cases of perforation in the first portion in which the situation of the perforation was noted, 68 were on the anterior wall, 39 on the posterior, 10 on the superior, and 1 on the inferior. In 8 perforations of the second portion, 5 were on the internal wall, 2 on the posterior, and 1 on the external. In 14 cases of perforated duodenal ulcer reported by Mr. Maynard Smith, 13 were in the first part of the duodenum, and of these 8 were noted to be on the anterior wall.

Number.—Usually one ulcer only is found (86 per cent); occasionally there may be two (9 per cent), or more (5 per cent), and if so they are generally found in different portions of the duodenum and are almost invariably acute.

In *form* and *dimension* they resemble the gastric ulcer; they are generally circular, but may be oval or elliptical, especially when of recent origin. The chronic ulcer is larger and more irregular in form, occasionally it is annular, involving the greater portion of the circumference of the bowel.

Appearance.—The ulcer has the same appearance as the gastric ulcer, when recent it has the punched-out character; but in the chronic form the edges of the ulcer are thickened, irregular, everted, or undermined, often terraced and sloping, giving the ulcer a funnel shape. The floor is formed by the submucous or muscular coat, and in some few cases by the liver, pancreas, or gall-bladder, as adhesions between the duodenum and the neighbouring organs may arise. Occasionally the neighbouring portion of the mucous membrane shews polypoid excrescences.

The microscopic examination of the ulcer shews the same absence of inflammatory changes at the borders as is the case in the gastric ulcer.

Complications and Sequels.—These are similar to those met with in ulcer of the stomach.

Cicatrisation.—According to Sir C. Perry and Dr. Shaw cicatrisation occurs in only about 11 per cent of all cases; other authors as Krauss also speak of the rarity of cicatrised duodenal ulcers; this relative rarity is, however, only apparent, as probably many such cicatrices are overlooked. Cicatrisation may give rise to varying degrees of duodenal stenosis, leading most frequently to dilatation of the stomach, as the stenosis is usually close to the pylorus, or to dilatation of the duodenum when the stenosis is lower down. In 262 cases Collin noted dilatation of the stomach in 18 and dilatation of the duodenum in 4. Biermer records a case in which obliteration of the lumen of the duodenum was followed by dilatation of the oesophagus. Cicatrices situated in the vicinity of the biliary papilla may produce occlusion, more or less complete, of the common bile-duct with obstructive jaundice, and of the pancreatic duct with consecutive atrophy of the pancreas. Herzfelder relates an instance of rupture of the gall-bladder following extreme distension from this cause. Dr. French recorded thrombosis of the portal vein from compression of this vessel as a direct result of deep cicatrisation of a duodenal ulcer.

Diverticula.—This deformity was noted by the Fenwicks in 6 per cent of their cases of chronic ulcer. The diverticula usually occur opposite the ulcer, or between it and the pylorus, occasionally the base of the ulcer itself is evaginated, and more rarely a traction diverticulum is produced by the contraction of adhesions between the base of the ulcer and the liver, diaphragm, or colon (Perry and Shaw).

Erosion of Blood-vessels.—As most of the large arteries supplying the duodenum lie behind it or along its inner side, it is chiefly deep ulcers on the posterior wall that give rise to severe haemorrhage. Fatal haemorrhage occurs in 35 (the Fenwicks) or 33 per cent (Krauss). With regard to the source of the haemorrhage the Fenwicks noted among 15 cases that the pancreatico-duodenal was eroded in 10, the gastro-duodenal in 3, the pancreatic in 1, and a branch of the splenic in 1. Collin in his statistics gives the seat of haemorrhage as from the pancreatico-duodenal artery in 12, the right gastro-epiploic artery in 3, the pancreatic artery in 2, the hepatic artery in 1, the aorta in 2, the portal vein in 2, and the superior mesenteric vein in 2. Among other vessels which have been found eroded are the pyloric and coronary arteries and the inferior vena cava. Occasionally a small aneurysmal dilatation is found on the vessel at the seat of rupture (Perry and Shaw).

Perforation.—Perforation of the duodenum leading to general peritonitis has been observed in 61 out of 124 fatal cases (the Fenwicks), in 125 out of 262 cases (Collin). Chvostek, however, estimates the frequency of perforation at about 42 per cent of all cases. The several positions of the perforation and their relative frequency have already been considered. Since perforation occurs in the vast majority of cases on the anterior surface of the first part of the duodenum, the stomach contents are free to pass into the general peritoneal cavity. Clinical experience and

recently direct experiment on the human cadaver (Maynard Smith) have shewn that the extravasated gastric contents almost invariably pass first into the right kidney pouch, and thence along the outer side of the ascending colon to the pelvic brim, and over this into the pelvis, or from the kidney pouch across the upper part of the ascending colon, then downwards along its inner side, across the terminal portion of the ileum, and so into the pelvis. When, however, the perforation is preceded by the formation of adhesions to adjacent viscera, or is very minute, and the escape of contents is so small as to allow of the formation of delimiting adhesions, or when it occurs in the extraperitoneal portion of the duodenum, it leads to the formation of a localised abscess, which may eventually rupture into the general peritoneal cavity, or to ulceration of some other viscus. In 12 cases the abscess was localised by adhesions between the liver, stomach, intestines, and abdominal wall in the vicinity of the perforation. In 2 it was subphrenic, one to the right, the other to the left, of the falciform ligament; in 6 it was situated in the retroperitoneal tissue, in 5 of these cases the pus tracked downwards and presented in one or other iliac fossa, and in one it appears to have ascended along the posterior mediastinum and to have pointed in the neck. In other cases the pus has been known to penetrate through the muscles of the back and point posteriorly near the spinal column or track anteriorly and point on the chest wall. In 2 cases ulceration extended into the pancreas and set up an abscess. Other instances are recorded of perforation into the gall-bladder, aorta (2 cases, Stich), portal vein (2 cases, Rayer, Habershon), superior mesenteric vein (Warfinger), hepatic artery (Vonwyl), and into the adjacent colon with the formation of a bimucous fistula (2 cases, Murchison, Saunderson). It may be the seat of cancerous growth, though not nearly so frequently as in the case of gastric ulcer; at least 10 examples of this are on record (*vide* p. 578).

Pathogenesis.—The character of duodenal ulcer is the same as that of ulcer of the stomach, and its formation is due to the same cause or causes. That the gastric juice plays an important part in its formation is shewn by the occurrence of the ulcer—in by far the majority of cases—in the first part of the duodenum, where the acidity of the gastric juice is not as yet neutralised by the alkaline pancreatic juice. It is highly probable also that a disturbance in the circulation, leading to a localised haemorrhagic infiltration similar to that observed in ulcers of the stomach, occurs as the early stage; and probably the same causes operate here as were duly considered in the pathology of gastric ulcer.

Symptomatology.—Anatomically we may distinguish the acute and chronic ulcers of the duodenum, but clinically this distinction can scarcely be made with any certainty; for though the first characteristic symptoms may be acute, such as profuse haemorrhage or signs of perforation, yet the ulcer may have existed for a long time previously without having given rise to any definite symptom. Leaving this distinction out of sight, we may divide cases of duodenal ulcer from the clinical point of view into certain groups, namely, those in which the course is chronic

and in which symptoms enable us to diagnose the duodenal ulcer; those in which the first symptoms are due either to excessive haemorrhage (melaena with or without haematemesis) or to perforation; and those in which signs of stenosis of the duodenum or of the common bile-duct form the prominent feature. In many cases, however, the symptoms are so indefinite that no diagnosis can be made.

Latency.—An ulcer of the duodenum may be entirely latent; scars of healed duodenal ulcers are sometimes found in subjects who have never had any manifestations of such a lesion, or in other cases the disease may run its course without symptoms until perforation or a severe haemorrhage leads to its discovery. Thus, in 60 per cent of the cases collected by Sir C. Perry and Dr. Shaw there were no symptoms of importance prior to the onset of the fatal perforation or haemorrhage, and in 53 per cent of those analysed by Cullen there was a similar latency. The Fenwicks, differentiating the acute and chronic forms of the lesion, found that 74 per cent of their chronic cases had characteristic symptoms before death. Krauss estimates that about one-fifth of all cases observed are latent. Ulcers close to the pylorus cannot be distinguished from gastric ulcers, though it is asserted that in these cases the localised pain is situated more deeply and to the right of the epigastric region, and that melaena occurs before the haematemesis; but these signs may be present in gastric ulcer.

Considering the cases of our first group, the chief symptoms which indicate the existence of a duodenal ulcer are deep-seated and localised pain occurring several hours after food, dyspepsia, and haemorrhage.

The pain varies in intensity: sometimes it is paroxysmal and agonising; at other times the patient only complains of a dull ache, a sense of uneasiness, or of painful distension. The pain is situated to the right and a little above the umbilicus; it radiates thence to the epigastrium, round to the right side, or to the umbilicus (the umbilical pain is most likely the associated pain in affection of the duodenum, but as it is also associated with affection of other portions of the small intestine, it is of no great diagnostic importance). The pain may radiate into the caecal region or along the right genito-crural nerve, simulating renal pain. It never passes to the right subscapular region as hepatic pain does. Localisation of the pain to a well-defined area is rarely observed, nor is it usually present over the dorsal vertebrae; it is generally not a continuous pain, but in its most characteristic form occurs from two and a half to four hours after meals, and lasts for some time; it is increased by pressure. According to Chvostek the time of its appearance after food varies with the quantity and quality of the food; it occurs earlier after breakfast, later after a more copious meal; and both he and Boas noticed that the pain stopped, at least for a time, if the patient was given a little wine or milk, which is explained by the wine causing reflex closure of the pylorus, preventing further egress of stomach contents into the duodenum. According to Johnston vomiting does not relieve the pain as it does in gastric ulcer, but Nothnagel

regarded this as very uncertain. In the majority of the Fenwicks' cases the onset of the pain was quite independent of food, and was often most severe when the stomach was empty. Occasionally it was excited by exertion or by some expiratory effort, such as laughing, coughing, or sneezing. Corresponding to the situation of the pain, there is generally an area of tenderness, especially when the ulcer is situated on the anterior or outer wall. Rigidity of the right rectus muscle is also a frequent phenomenon during the attacks of pain.

Haemorrhage of a severe type occurs in from 30 to 40 per cent of all cases. The blood may be vomited, passed by the bowels, or be evacuated in both ways. In 17 cases the Fenwicks noted haematemesis alone in 5, melaena alone in 7, and both haematemesis and melaena in 5. In 34 cases observed by Oppenheimer there was haematemesis in 8, melaena in 10, and both haematemesis and melaena in 16. The haemorrhage may be so slight as not to be perceived by the patient (occult haemorrhage), or profuse, occurring only once, or several times in a short space of time. The patient may feel a colicky pain, and immediately a desire to go to stool; or he may have a sudden feeling of faintness, accompanied by vomiting and loss of consciousness; or, in very severe cases (of which I have seen one example, the diagnosis being verified by autopsy), he may have convulsions. As the patient is not always conscious of having passed blood, and as sometimes the discharge of blood may not occur till some time after the above symptoms have appeared, it is always well in cases of sudden faintness, marked by anaemia, in which dyspeptic symptoms have existed for some time, to examine the stools. The *dyspeptic symptoms* are variable; often the appetite is normal; the patient may complain of a fulness and heaviness coming on some hours after a meal; he often also complains of acidity, water-brash, and flatulence; vomiting is rare, it may be altogether absent, or occur at long intervals; when it does occur it is reflex, depending on the severity of the pain. The gastric contents vary as regards their hydrochloric acid content; in some cases it has been absent, in some normal, and in others increased. When cicatrices have produced duodenal stenosis the vomit is like that of a dilated stomach. There is more often constipation than diarrhoea. The health of the patient suffers at first but little; after a time there is emaciation. If the attacks of melaena occur often, or if a large quantity of blood be discharged, the patient becomes very anaemic and weak.

In the second group of cases, the first and chief symptoms for which we are called upon to treat the patient are either those of profuse haemorrhage or of perforation. If haemorrhage in the form of melaena be the first symptom, we have to inquire into the history and the presence of localised pain in the abdomen, as melaena may occur in other affections such as cirrhosis of the liver, cancer of the intestines, chronic valvular affection of the heart, dysentery, pernicious anaemia, purpura, and so forth. It is not until we have eliminated these affections that the diagnosis of duodenal ulcer can be made with some degree of certainty. The symptoms are those of any internal haemorrhage, and need not be

described again. In some cases the melaena disappears after a few days; the patient remains anaemic and weak for some time and then gradually recovers: in other cases the patient may die from syncope or collapse. If haematemesis be the first symptom, as not uncommonly happens, then differentiation from gastric ulcer will have to be considered on the lines already laid down (p. 478). The mortality from haemorrhage is about one-third of all cases; in the Fenwicks' series it was 36 per cent, in Cullen's 20 to 30 per cent, while in Sir C. Perry and Dr. Shaw's cases it accounted for 13 per cent only.

Perforation of a latent duodenal ulcer produces the same symptoms as those described when treating of perforation of a gastric ulcer; with perhaps this difference, however, that vomiting may be more persistent. Pain in the right iliac fossa, due to peritonitis set up by fluid flowing to that region (*vide* p. 560), occurs in cases of perforation of a duodenal ulcer as in those of gastric ulcer, and may lead to a diagnosis of perforation of the appendix. In the 51 cases collected by Mr. Moynihan 19 were diagnosed as appendicitis. On the other hand, it is important to remember that occasionally perforative appendicitis may simulate gastric or duodenal perforation, and also that the two conditions may coexist, of which there are already some six instances on record (Carter, Lediard and Sedgwick, Watson Cheyne, Low, Smith).

In the third group of cases an ulcer, not having itself given rise to any definite symptoms, leads to narrowing of the duodenum, and causes symptoms similar to those of stenosis of the pylorus; or an ulcer, situated over the outlet of the common bile-duct, by its cicatrisation causes persistent jaundice from occlusion of the duct. The first class of cases may be indistinguishable from stenosis of the pylorus: in other cases, however, the examination of the gastric juice or of the vomit will, it has been said, shew that the obstruction is situated below the biliary papilla, for the gastric juice will have an alkaline reaction, will contain bile, and will digest fibrin (Boas).

Stenosis of the bile-duct due to duodenal ulcer will in most cases be indistinguishable from the symptoms produced by an impacted gall-stone; occasionally, however, a correct diagnosis is possible. In such cases (Perry and Shaw) the jaundice has become very intense and the patients much emaciated, the interval from the onset to the fatal termination varying from some months to some years.

The treatment of duodenal ulcer and its complications is the same as that of gastric ulcer.

Surgical treatment is indicated when perforation occurs, and may be carried out when a subphrenic abscess arises, occasionally when there is stenosis of the duodenum, and in those cases in which the common bile-duct is narrowed or obliterated. In the latter cases cholecystenterostomy may accomplish a complete cure. This occurred in a patient under the care of my colleague, the late Mr. Thomas Jones, at the Manchester Infirmary; jaundice had existed for several months, and there was a narrowing of the common bile-duct, due either to ulcer or gall-

stone. Other similar cases are recorded by English and Continental observers.

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J. D.

DISEASES OF THE SMALL INTESTINE

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UNDER this heading reference will be made to those morbid conditions only which have not been described elsewhere, lest any be omitted from the *System of Medicine*. In many instances the lesions of the small intestine have been described under other headings, such as enteric fever, intestinal obstruction, and so forth ; and a cross reference is all that is required.

SYNOPSIS

Malformations.—Occlusion and stenosis of the duodenum ; of the small intestine ; diverticula ; duodenal pouches.

Vascular disturbances of intestine : phlegmonous enteritis.

Lardaceous disease.

Ulceration : tuberculous, syphilitic, uraemic, peptic jejunal.

Innocent tumours (*a*) in the duodenum ; (*b*) in the jejunum and ileum.

Lymphadenoma.

Malignant growths.—Primary : (*a*) in the duodenum ; (*b*) in the jejunum and ileum. Secondary.

MALFORMATIONS. — **Congenital occlusion and stenosis of the duodenum** are usually situated close to, and commonly just above,

the biliary papilla. Complete occlusion is much commoner than stenosis; in 57 cases collected by Cordes there were 48 of occlusion and 9 of stenosis. There may be a diaphragm, with or without a perforation in the centre, running across the bowel, or the continuity of the bowel may be completely interrupted. The septum is composed of the muscular as well as of the mucous and submucous coats of the bowel; its position is constant, and points to the conclusion that it is connected with the budding-off of the hepatic diverticula in foetal life. Mr. Shattock explains the mechanism both of this congenital defect and of the analogous one of congenital atresia of the oesophagus, by supposing that the unequal growth which leads to the formation of the glandular diverticula (liver or lungs) produces a kink on the opposite side of the original tube, much as will happen if the intestine be drawn out in an artificial anus through any thickness of the abdominal wall, and that this displacement may conceivably lead to secondary closure of the original tube. In 11 out of Cordes' 57 cases there was some other malformation, in one case atresia of both the oesophagus and rectum.

The stomach and the part of the duodenum above the occlusion become dilated, and may form a tense bilocular cyst suggesting a double stomach; this part of the duodenum may end blindly as a cul-de-sac, or, as is generally the case, remain connected with the third part of the duodenum. The dilatation spreads from the first part of the duodenum to the stomach.

If the obstruction be complete, vomiting begins soon after birth and continues until death, which usually follows within five days. The complete absence of bile from the vomit should suggest this form of congenital occlusion rather than one lower down, below the entrance of the bile-duct. The vomit may be clear at first, and subsequently of coffee-ground character. Meconium passes into the faeces, and may be voided per rectum. Jaundice is rare, being mentioned in 8 of Cordes' 57 cases. When the obstruction is not complete some food may pass through into the intestine, and life is not so rapidly brought to a close; Lardennois reported a case of narrowing of the first part of the duodenum, regarded as congenital, in a man aged thirty-six years.

Congenital narrowing, obliteration, or complete interruption of the alimentary canal may occur at the junction of the duodenum with the jejunum. Ducros collected three cases of stenosis, five of obliteration of the lumen, and three of complete interruption of the intestine at this point. The probable cause of such changes in this situation is pressure from the anatomical arrangement of the peritoneum at the duodeno-jejunal junction, and the condition thus resembles stricture of the third part of the duodenum produced in adults, often the subjects of visceroptosis, by the pressure of the mesenteric vessels (*vide* p. 768), of which Conner has collected 18 cases.

The second part of the duodenum may be encircled by a ring of pancreatic tissue; according to Mr. Mayo Robson at least 6 cases of this kind are on record, and in the event of inflammation supervening in the

pancreatic tissue, narrowing of the duodenum with symptoms resembling those of pyloric obstruction will result. This congenital abnormality may therefore dispose to duodenal stenosis.

Congenital Occlusion and Stenosis of the Small Intestine.—In occlusion the intestine may be interrupted, and the intestinal walls only represented by a cord connecting the pervious parts of the bowel. These occlusions may be multiple. They are of course incompatible with life for more than a few days. In other instances the small intestine may be stenosed by contraction of its walls, or may be obstructed by a diaphragm stretched across its lumen like that seen in the duodenum. This narrowing is most frequent in the lower part of the ileum, in the situation, that is, where the diverticulum ilei occurs. Sometimes a fibrous band representing the remains of the vitelline duct is seen to be attached at the point of the stricture; in such cases the stenosis might be mechanical and due to the intestine winding the band round itself; but such cases are exceptional. More often the stenosis is unaccompanied by any remains of the vitelline duct.

The most probable explanation of the presence of single obliterations, narrowings, or diaphragmatic membranes in the ileum is that the process of atrophy and involution of the vitelline duct has become excessive, and by spreading to the intestine at the point of junction has produced similar changes there.

In other cases similar but multiple narrowings of the small intestine are found. They shew no sign of past ulceration or other morbid process, and though less advanced are of the same pattern as the multiple complete obliterations mentioned above, and are presumably due to the same cause. Whatever this may be, it is active in foetal life, and cannot have anything to do with obliteration of the vitelline duct. Intra-uterine peritonitis, by interfering early in foetal life with the proper development of the intestine, or later by producing bands and adhesions by which the intestine is constricted and locally obliterated, offers a possible explanation. While abnormal development of the valvulae conniventes has been suggested as an explanation of multiple diaphragmatic strictures, the cause of this hypertrophy of the valvulae conniventes is wanting. It is rather against this view that the valvulae conniventes are but slightly developed in foetal life. (See also pp. 572, 760.)

Clinically, partial single narrowing of the lumen, such as may occur in the ileum near the site of Meckel's diverticulum, need not give rise to any signs of obstruction, and is compatible with long life. I have seen a case with three diaphragmatic obstructions in a man aged twenty-five years without any intestinal troubles. In other cases there may be signs of chronic obstruction. The liquid state of the faeces in the small intestine accounts for the absence of symptoms when there is a considerable amount of narrowing of the calibre of the intestine.

Diverticula of the small intestine fall into two distinct groups :—

(*a*) *Congenital*, which are true or composed of all the coats of the

intestine. The only important or common congenital diverticulum is Meckel's diverticulum, which is fully described in the article on "Intestinal Obstruction" (*vide* p. 771). A few examples of congenital diverticula with a small accessory pancreas attached to the tip have been recorded; it has been thought that the diverticula were due to traction exerted by the accessory pancreas.

(*b*) *Acquired* diverticula of the small intestine are probably always false or devoid of muscular fibres, though possibly a few isolated muscular fibres may occasionally stray over the pouches. They are hernial protrusions of the mucosa through the muscular coats, and are found close to the attachment and often between the layers of the mesentery. They arise in connexion with the passage of the veins through the muscular coats of the intestine, and depend on weakness of the intestinal wall in this situation. They are more often met with in elderly subjects; Hansemann was unable to produce them experimentally in young subjects, but succeeded in senile bodies. They are probably favoured by increased pressure inside the intestine. They are usually numerous; as many as 400 have been recorded (Hansemann). They vary in size from that of a pea to that of a hen's egg. The peritoneum covering them may be thickened. Inflammation of these pouches (diverticulitis) has been known to spread to the mesentery, and by producing cicatrisation to lead to kinking or stenosis of the intestine. But results of this kind are very rare in connexion with the small intestine as compared with the colon, and as a rule no symptoms are connected with these diverticula.

Duodenal Pouches.—Pouches of small size, rarely more than three-quarters of an inch deep, are not uncommon in connexion with the biliary papilla. They are false and do not contain any muscle in their walls. From their position they have been thought to be congenital, and to depend on weakening of the wall of the duodenum by the primitive hepatic diverticula. It is, however, probable that they are acquired, and the result of enteroptosis. Dr. Keith states that they are present in all women whose bodies shew evidence that they have worn corsets, and that the duodenum, being depressed downwards, is held up at one spot by the bile-duct, which, being surrounded with firm fibrous tissue, does not give, and pulls the mucous membrane out into a pouch. Usually the pouches are single, but they may be bifid (Nattan-Larrier, Rolleston and Fenton). A similar pouch has been seen at the opening of Santorini's accessory duct of the pancreas. No symptoms or evil results are known to be due to duodenal pouches; it is conceivable that, if filled with food, they might exert pressure on the common bile-duct. Pertik records a glove-shaped diverticulum which, as it became filled with food, must have exerted pressure on the lumen of the duodenum and narrowed it; but this is probably a unique case.

It is only necessary to mention very briefly other forms of local dilatation or pouching of the intestinal wall in which all the coats are represented. The traction of adhesions may in rare instances induce a

traction diverticulum. Infiltration of the wall of the intestine by a sarcoma may produce a marked dilatation. Foreign bodies, such as gall-stones, may, by inducing local inflammatory changes, lead to a pouching of the intestinal wall. A local bulging may be due to the damage inflicted in a strangulated hernia, and the effects of the intestinal paralysis consequent on such conditions may be manifest, even after the lapse of years, in want of tone and contractility (Barker).

Vascular disturbances in the intestines are of very various kinds. —The chronic venous engorgement of portal stagnation, whether this be due to tricuspid regurgitation or to obstruction in the liver as in cirrhosis, easily passes into catarrhal inflammation, and is then characterised by the presence of tenacious mucus on the surface of the intestine. Localised dilatation of veins is sometimes seen in this condition, but may occur independently.

Active congestion may, of course, be seen post-mortem in toxic enteritis and diarrhoea, but it is frequently absent when, from clinical evidence, it might have been expected.

Haemorrhages into the substance of the intestinal mucous membrane or into the submucous coat occur in infective endocarditis and pyaemia from embolism, sometimes in thrombosis, in purpura, in which ulceration has also been noted, in strangulation of the bowel, and in other cases in which the cause may not be evident. In the last case the haemorrhage may be extensive and suggest embolism or thrombosis of the mesenteric vessels, but careful search may fail to bring such a cause to light; some of these cases seem to be allied to the sanguineous peritoneal cysts. Embolism and thrombosis of the mesenteric vessels are dealt with in detail elsewhere (Vol. VI.); haemorrhagic infarction of the intestine results, and is followed by symptoms which may be either acute and mainly those of acute obstruction, with passage of blood by the bowel, or chronic and less definite.

Phlegmonous enteritis is a very rare condition, and is so little known that it is probably often not recognised when it does occur; thus, since reading MacCallum's article on the subject I can recall two examples, both in the duodenum and imitating intestinal obstruction so that laparotomy was performed, which were regarded as an acute form of enteritis but not further identified. Of MacCallum's 7 collected cases 5 were in the upper part of the small intestine or in the duodenum, 1 in the colon, and 1 implicated the whole of the digestive tract. There is some reason to believe that the relatively fixed position of the duodenum plays a part by favouring damage to the coats of the bowel from traumatism, and so enabling infection to occur. The affected segment of the intestine presents a remarkable appearance, being much enlarged and forming a rigid tube with oedematous walls. The mucous membrane is usually little affected, but may be oedematous or ulcerated in places; the submucous and muscular coats are swollen from infiltration with inflammatory cells and oedema, and may exude pus; streptococci have been found. The

condition is analogous to phlegmonous gastritis. The symptoms are vomiting, constipation, distension, and suggest intestinal obstruction. The condition can hardly be diagnosed before the abdomen is opened; the prognosis is extremely grave. MacCallum suggests enterostomy to relieve the symptoms of obstruction.

Lardaceous Disease of the Intestine.—The alimentary canal is relatively often affected in cases of lardaceous disease, coming fourth among the organs—after the kidneys, spleen, and liver—liable to this change. Since the forms of prolonged suppuration amenable to surgical treatment have now become so comparatively rare, syphilis and pulmonary tuberculosis now occupy a more important place in the etiology of lardaceous change than they did in pre-antiseptic times. (For "Pathology of Lardaceous Change" see Vol. I. p. 570.) The whole of the alimentary canal may be affected or the change may be partial, and then usually falls on the small intestine and especially on the ileum. Lardaceous disease and tuberculous ulceration of the intestine are not infrequently associated. The clinical manifestation of lardaceous disease of the intestine is diarrhoea, which is probably due to imperfect absorption of the watery constituents of the intestinal contents and not to excessive secretion or to increased peristalsis as in most other forms of diarrhoea. The motions, which are passed without pain, are watery, but do not present any special characters. It has been stated that the intestinal villi, being specially fragile from the lardaceous change, are prone to break off and so to give rise to haemorrhage; but it is generally agreed that, unless complicated by tuberculous or some other form of ulceration, lardaceous disease of the intestine is not characterised by the presence of blood, at any rate manifest to the naked eye, in the stools. The diarrhoea is obstinate and, though less frequent, is probably a more serious result of lardaceous disease than albuminuria, as it is the direct cause of death in a considerable proportion of cases of lardaceous disease. The prognosis is bad. Treatment should be directed to removal or alleviation of the underlying cause by surgical measures or by antisyphilitic treatment, and the general health should be maintained by healthy surroundings and suitable food. The diarrhoea is usually treated by various preparations of bismuth and opium.

Ulceration of the intestines is described under so many other headings, that it would weary the reader, and, indeed, be unnecessary, to give all the cross references to the diseases of which ulceration of the small intestine is either an integral part or a more or less direct result. Generally speaking, reference should be made to the articles on "Diarrhoea," "Diarrhoea in Children."

Tuberculous Ulceration.—*Modes of Attack.*—In generalised tuberculosis, as in other parts of the body, miliary tubercles may occur in the mucosa of the intestine, but the deposit is, of course, of no clinical importance.

Primary infection of the mucous membrane from tuberculous milk or

meat contaminated with tuberculous material, or even from butter, occurs very rarely in adults; more commonly in children.

Secondary tuberculosis of the intestine due to the swallowing of sputum containing tubercle bacilli is very common, and has been found in over 50 per cent of fatal cases of pulmonary tuberculosis. It must be borne in mind, however, that tuberculous patients may suffer from follicular ulceration of the small intestine, due to the pyococci contained in the sputum they swallow; and that every ulcer found in the small intestine of a patient dying from tuberculosis is not necessarily tuberculous. The presence of simple follicular ulcers certainly renders the intestine more vulnerable to tuberculous infection, and a secondary infection may take place. Chronic intestinal catarrh, such as that seen in rickets and alcoholism, is probably no inconsiderable disposing factor to the development of intestinal tuberculosis.

Infection of the mucous membrane of the intestine may possibly occur by the extension of the tuberculous process from the peritoneum, or as the result of the discharge of tuberculous glands or abscesses into the lumen of the intestine; but these modes of infection are quite exceptional.

Situation.—Tuberculous ulceration of the intestine is commonest in the lower part of the ileum, and though the ulcers may extend throughout the whole of the small intestine, they become less frequent as the duodenum is approached, where they are very rare; ulceration has been seen, however, within half an inch of the pylorus. Since it is thought that the acidity of the gastric juice tends to prevent the occurrence of tuberculous ulceration, it is probable that in such exceptional instances this secretion was deficient in hydrochloric acid. The ulcers, like those of enteric fever, are more common around the ileo-caecal valve, and may extend into the appendix, giving rise to symptoms of appendicitis, or into the colon. The colon may indeed be affected without the ileum being attacked, while the rectum is a not infrequent situation for such tuberculous ulcers.

Anatomical Characters.—The tubercles generally begin either in Peyer's patches or in the solitary glands, and after undergoing caseation appear as small yellowish spots, which soften down in the centre, run into each other, and by opening into the intestine give rise to the earliest stage of a tuberculous ulcer. The lymphoid and other tissues around are swollen from extension of the inflammatory process, and the edges of the ulcer are raised. The ulcer extends by softening and discharge of the surrounding tubercles, while at the same time infiltration of the coats of the intestine is proceeding. The lymphatic vessels become affected, and local chronic peritonitis is set up. The base of the ulcer thus becomes thickened by tuberculous inflammation in its muscular and serous walls, and small white or even yellow tubercles can be seen on the peritoneal surface, which appears somewhat opaque and thickened. The base of the ulcer is thus protected against perforation, and may be considerably thicker than the healthy parts of the intestine. The danger of perforation is still further prevented by the tendency of the local tuberculous peritonitis to cause

adhesions to surrounding coils of intestine. In some cases these adhesions may be drawn out into bands (*vide* article on "Intestinal Obstruction," p. 770) or may be broken across; they are then seen as filamentous tags on the outside of the intestine, and become more evident when the intestine is placed in water. The chronic inflammation may give rise to pigmentation of the peritoneal or of the intestinal surface of the ulcer; this is more often seen in the chronic oblong ulcers in the colon. The thickened base of the ulcer is usually roughened and irregular; sloughs are only present exceptionally. The edges of the ulcer are raised, thickened, and not undermined. The ulcers, which in the early stage are small and round, become large and irregular in outline; like all chronic ulcers they are apt to run transversely round the lumen of the bowel, this extension being preceded by tuberculous infection of the lymphatic vessels, the circular course of which is shewn up by caseous spots. In addition, the mucous membrane may be lardaceous. The histological appearances of tuberculosis are fully described in the article on "Tuberculosis" (Vol. II. Part I. p. 263).

Hyperplastic tuberculosis of the ileo-caecal region is a remarkable condition (*vide* p. 760) which imitates a new growth both in the great thickening of the intestinal wall and in producing stenosis. It is very chronic, and has been thought to be due to infection with attenuated tubercle bacilli and other organisms, a mixture of tuberculous and simple inflammatory lesions resulting (Lartigau), or to bovine tuberculosis. The caecum and ileo-caecal valve are the usual situations, and the change may extend for a short distance into the ileum. In rare instances the change is confined to the small intestine [Pantaloni, Michon]. In Michon's case appendicitis was diagnosed.

Results of Tuberculous Ulceration.—Perforation is rare, and after death it is often very difficult to be certain whether perforation of an ulcer into the peritoneal cavity took place during life or not. For with tuberculous enteritis may be combined peritonitis, and matting together of the intestines, with loculi containing ascitic fluid turbid with varying amounts of fibrin or pus-cells; and the intestines are so softened that any attempt to ascertain whether there be a perforation of an ulcer into the cavity of the peritoneum readily leads to rupture. In some cases it may be that perforation into the intestine from without has taken place either from a localised collection of pus or from a softened caseous lymphatic gland, and not in the reverse direction. The inflamed walls of the intestinal ulcers, under the conditions such as are present when there is advanced tuberculous peritonitis, may allow of the passage of micro-organisms, such as *B. coli*, without any solution of continuity; and thus a more acute peritonitis, due to secondary infection, is set up. Though this is not a gross perforation of the intestine it comes eventually to much the same thing; it leads, that is, to increased severity of the peritoneal inflammation. Perforation does occur, but it is rare, and contrasts with what holds in acute intestinal ulceration, as in enteric fever.

Stricture of the small intestine, from healing of the ulcers which run

round the lumen of the intestine, occurs more frequently than is generally thought. Probably many so-called simple strictures are in reality of tuberculous origin. Bernay has collected 70 recorded cases. According to Tuffier, there are three forms of tuberculous stricture of the small intestine: (*a*) due to cicatrisation of an ulcer; (*b*) due to contraction of tuberculous granulation-tissue without any ulceration; this leads to a diaphragmatic stricture; and (*c*) hyperplastic tuberculosis. But the comparatively slight narrowing that results and the fluid state of the intestinal contents combine to render signs of obstruction from tuberculous strictures, apart from the effect of tuberculous peritonitis, rare. The strictures may be multiple; in a case of Nothnagel's there were seven strictures in the small intestine (*vide* p. 760).

Symptoms.—Very frequently no symptoms are present during life, even when, as seen after death, there is very considerable tuberculous ulceration in the small intestine; though from their occurrence in more than 50 per cent of the fatal cases of pulmonary tuberculosis their existence may be suspected whenever, in the course of the disease, there is diarrhoea lasting for any time, abdominal tenderness, or pain. The diarrhoea may, though rarely, be accompanied by blood. Prof. Osler has seen several cases of fatal intestinal haemorrhage, but this must be most exceptional. It must be remembered that simple diarrhoea in pulmonary tuberculosis may be due to lardaceous disease. The presence or absence of diarrhoea seems to depend chiefly on ulceration of the colon, since it may be absent when the ileum is extensively affected. Generally speaking, however, diarrhoea in pulmonary tuberculosis indicates tuberculous ulceration.

Diagnosis.—An important factor, as already mentioned, is the existence of tuberculous disease of the lungs or peritoneum. The passage of blood, when it occurs, would distinguish it from lardaceous disease, which is very rarely accompanied by simple ulceration of the intestine. The presence of tubercle bacilli in the faeces would of course be strong evidence, but little value could be attached to a negative result.

In children, in whom primary tuberculous disease of the intestine may occur without evidence of pulmonary disease, the diagnosis between gastro-intestinal catarrh and tuberculous ulceration is less easy than in adults. The presence of fever, abdominal distension, enlarged glands, and wasting and debility out of proportion to the diarrhoea, together with the absence of worms and of any other cause for the condition, are in favour of tuberculous ulceration.

Treatment.—In adults tuberculous ulceration is nearly always secondary to pulmonary tuberculosis, and the lines of treatment will be found in the special article on that disease (Vol. V.). It is important to guard against setting up intestinal catarrh, and so bringing on diarrhoea which may prove very rebellious to treatment. Strong purgatives may have this effect, so that care is required in combating constipation in pulmonary tuberculosis. Food of a bland, nutritious character, which can easily be digested and absorbed, is important; if need be, it may be predigested. Milk and eggs can usually be borne, but if milk be not absorbed, as

shewn by the presence of curds in the stools, it should be diminished in quantity or omitted for a time from the dietary. Pounded fish or meat in small quantities at a time, chicken broth and soups thickened with farinaceous preparations, Mellin's food, and gruel are usually suitable.

The healing of the ulcers will be impeded by intestinal fermentation or by any irritating form of food, and it is probable that creasote and the allied drugs exert both a preventive and curative influence on intestinal ulceration. Besides the observance of careful feeding, it is important to avoid any such exciting factors as exposure to wet or cold. When diarrhoea occurs it should be controlled by opium, chalk, bismuth, and astringents, and if necessary by morphia suppositories, or enemas containing opium. The patient should be kept in bed, and warm applications to the abdomen may be tried.

Syphilitic Ulceration.—Manifestations of syphilitic infection, though comparatively common at the two ends of the alimentary canal, are very rarely recognised in the intervening portion. Mucous patches, pharyngeal lesions, condylomas, and stricture of the rectum are well known and frequently met with, and are described elsewhere in this *System*. But syphilitic ulceration in the intestine is rare.

In infantile syphilis gummas beginning in the submucous coat, and when large invading the muscular coat, have been described. Thickened plaques, often in Peyer's patches and breaking down and ulcerating, as well as minute gummas in the mucosa, have been found. The presence of undoubted syphilitic lesions elsewhere, such as gummas in other organs, the general histological resemblance of the intestinal lesions to those characterising syphilis, and the absence of any evidence that the lesions are tuberculous, point to their syphilitic nature. But they are rare. Peritonitis may result from their presence, and even perforation of the intestine. These lesions begin in the seventh month of intra-uterine life, and may be well developed in the eighth. In addition to the general symptoms of infantile syphilis diarrhoea and melaena may be present. The treatment is that of the general disease by mercury.

Syphilitic Lesions in Acquired Syphilis.—The affections of the pharynx and anus seem hardly ever to extend into the neighbouring part of the intestinal tract; and though individuals with secondary syphilis may present intestinal ulceration, there is little convincing evidence that it is syphilitic in origin.

In tertiary syphilis the intestine may become lardaceous, and in exceptional cases ulceration has supervened. Apart from the rectum and adjacent part of the colon, the question again arises whether in any case of ulceration we are dealing with syphilitic ulceration, or merely with ulceration in a syphilitic subject; in some of the recorded cases the latter alternative appears more than probable. In this connexion the not infrequent incidence of tuberculosis in syphilitic subjects should be borne in mind. Superficial necrotic ulcers, gummas, and multiple small syphilomas resembling tubercles may occur. The ulcers may perforate and set up general peritonitis, or may heal and produce strictures.

Gummas may obstruct the lumen of the intestine and give rise to intestinal obstruction or break down into ulcers. The treatment is of course that of syphilis.

Uraemic Ulcers.—Ulceration of the alimentary canal in fatal cases of chronic renal disease has long been recognised. In 1859 Treitz described these ulcers, and explained their presence as the result of irritation due to carbonate of ammonia. In this country Dr. Dickinson recorded 22 cases under the name of albuminuric ulcers, and believed that they were due to haemorrhage into the submucous coat; this explanation has not met with universal acceptance (cf. p. 838), and other hypotheses have been put forward. Nothnagel inclined to the opinion that poisons in the blood excreted into the intestine were responsible for the ulcers, and in favour of this is the occasional localisation of these ulcers in the first part of the duodenum, 18 cases of which have been collected by Barié and Delaunay, thus resembling ulceration after burns, which is probably due to toxins carried by the blood-stream. Capillary thrombosis (Hlava), arteriosclerosis of the intestinal arteries (Mathieu and Roux), and the irritation set up by vigorous purgation have also been incriminated. The ulcers occur in the duodenum, small and large intestine, and rarely in the stomach. They may be present in both the small and large intestine in the same case, but more commonly are confined to one or other of these two parts, which are affected in about the same proportion. They may be small or occupy a considerable area. Mathieu and Roux record a continuously ulcerated area of 30 inches in length in the ileum. In the small intestine the ileum is usually attacked. The ulcers may have undermined edges, shew pigmentation, and perforate into the peritoneal cavity (Dickinson). The ulcers are associated with interstitial nephritis, arteriosclerosis, and high blood-pressure; a considerable number of the cases are in young subjects with contracted white kidneys.

In some cases, especially when there are a few ulcers in the ileum only, there are no intestinal symptoms. Diarrhoea is the chief manifestation, and is often associated with vomiting probably uraemic in origin. Peritonitis may occur without perforation of the intestine. Caution must be exercised in restraining the diarrhoea, as grave uraemic manifestations may follow suppression of what may be in part a vicarious excretion of toxic products by the bowel.

Lardaceous ulcers have been described, and are explained as the outcome of malnutrition of the intestinal mucosa, but they must be extremely rare. Ulceration due to other causes, such as tuberculosis, may of course be present in a lardaceous intestine.

Peptic jejunal ulcer following gastro-enterostomy is rare. It occurs close to the union of the stomach and intestine, and, as it is regarded as due to the entrance of hyperacid gastric juice into the intestine, has been spoken of as a peptic ulcer. It appears to be more likely to follow anterior than posterior gastro-enterostomy: out of 27 cases 20 followed some form of the anterior and 7 some form of the posterior operation (Gosset). It has been suggested that the anterior operation does not

drain the stomach so efficiently as the posterior method, and that hyperchlorhydria is not so completely relieved. Peptic jejunal ulcer has been produced by experimental gastro-enterostomy in dogs (Watts). When the ulcer perforates, the resulting peritonitis is usually circumscribed (20 out of 31 cases); but in about a quarter of the cases (8 out of 31) general peritonitis follows, and in a few instances (3 out of 31) perforation into the colon or stomach. Mr. Mayo Robson considers that when peptic ulcer follows posterior gastro-enterostomy general peritonitis is more likely to result from perforation than in the case of an ulcer following the anterior operation. This form of ulcer is almost exclusively met with in males; in 31 cases collected by Gosset there were 29 men and 2 women. The symptoms of jejunal ulcer are much the same as those of gastric ulcer. The burning pain in the epigastrium is usually severe, is temporarily relieved by taking food, but in an hour or two returns with greater severity. Vomiting is frequent.

Follicular ulcers occasionally occur, and resemble those described in the article on "Diseases of the Colon" (*vide* p. 835).

In *leukaemia* considerable enlargement of Peyer's patches may occur, and, especially in acute cases, ulceration, resembling that of enteric fever, may follow and even go on to perforation.

Actinomycosis of the intestine usually affects the appendix (*vide* p. 606); perhaps in some cases it begins in the ileum close to the appendix, and gives rise to much the same signs as in the appendicular form.

INNOCENT TUMOURS.—In the small intestine innocent tumours are usually small and multiple.

In the duodenum innocent tumours are rarer than in the rest of the small intestine. Polypoid adenomas are occasionally met with, usually in association with similar tumours elsewhere in the intestines; in Dr. W. Collier's case they were more numerous in the duodenum than elsewhere. In some cases a solitary papilloma arises in connexion with the biliary papilla, and may give rise to intermittent biliary obstruction and favour an ascending infection of the common bile-duct, thus behaving like a carcinoma in this position (*vide* p. 579). A large myxofibromatous polypus in the second part of the duodenum has been known to cause pyloric obstruction. Polyadenoma of Brunner's glands has been described by Besse and by de Rouville and Martin as a cause of duodenal ulcer. In conjunction with Dr. Trevor I have described a case of spheroidal-celled carcinoma starting from a polyadenoma of Brunner's glands associated with an old duodenal ulcer. In general lymphangiectasis of the intestinal mucosa the duodenum may be affected, and in one case the duodenum and jejunum shewed the most marked change (Allchin and Hebb). The Westminster Hospital Museum contains a cyst, lined with normal mucous membrane, in the posterior wall of the first part of the duodenum; during life it gave rise to symptoms of pyloric obstruction. A cyst, the size of a tangerine orange and containing bile, was described by Dr. Trevor; it projected into the duodenum in the situation of the biliary papilla, and was regarded as a congenital abnormality due to

fusion in the middle line of the folds of mucosa which normally enclose the fossa for the papilla. The patient, who died from a perforating duodenal ulcer, was not jaundiced.

In the **jejunum and ileum** various forms of innocent tumours occur, and, like innocent tumours generally, are not uncommonly multiple and very frequently pedunculated.

Adenomas, composed of the mucous and submucous coats of the intestine, are less often seen than in the colon and rectum. Occasionally a single one is found above a stricture, but when present they are generally multiple and due to developmental aberration. In extreme instances there have been thousands of those mucous polypi; this condition of multiple mucous polypi may, like some other forms of innocent growth, be hereditary. An important point about these multiple growths of the alimentary canal is that carcinoma may develop; this occurred in 20 of the 42 cases collected by Quénu and Landel, but apparently takes place in the colon only. Mr. Childe has recorded carcinoma of the rectum in three sisters, in two of whom it supervened on multiple adenomas. (For tumours of the umbilicus derived from the vitelline duct see p. 771.)

Myoma, fibroma, and fibromyoma may be either submucous or subserous. When submucous, 50 per cent of them give rise to intussusception; but they may obstruct the lumen without producing any invagination. Of 36 cases collected by Dewis, 17 (3 in the duodenum, 2 in the jejunum, 11 in the ileum, 1 undefined) were in the small intestine. *Lipomas* are occasionally seen in the small intestine. Dewis collected 17 (6 in the duodenum, 4 in the jejunum, and 7 in the ileum). A doubtful and unique case of osteoma of the small intestine has been reported by Quénu, and one of a hair-ball by Perman. Guy's Hospital Museum (No. 891) contains a polypus of the ileum with a central mass of fat, which was thought to be due to invagination of a Meckel's diverticulum taking some of the mesenteric fat in with it.

Innumerable villosities due to lymphangiectasis have been described, and in a minor degree dilatation of the lacteals is not so very rare, and has been found in association with chylous ascites. MacCallum has collected 6 examples of multiple *haemangiomas* of the small intestine; their presence may be associated with gastro-intestinal haemorrhage.

Multiple congenital *cysts* of the intestine have been explained on the hypothesis that foetal peritonitis cuts off small portions of the intestinal walls, and that these foetal inclusions subsequently become cystic from accumulation of mucus. Roth found an intestinal cyst lined with ciliated epithelium, an observation bearing in a suggestive manner on the character of the intestinal mucosa in early foetal life. In a man who died of pulmonary tuberculosis, Letulle found 300-400 cysts of various sizes in the submucous layer; the larger ones contained fluid, the smaller ones leucocytes and giant-cells, but as no tubercle bacilli could be found he did not regard them as tuberculous, but as derived from the mucosa. The enteroid or juxta-intestinal cysts, of which Terrier and Lecène have collected 18 examples, are situated near the lower end of

the ileum, and have been regarded as derived from the remains of Meckel's diverticulum. Clinically they resemble mesenteric cysts.

Lymphadenoma of the intestine is commonly recognised. Dr. Pitt describes two forms: (i.) lymphadenoma attacking the lymphoid tissue in the mucosa and submucosa, especially in the ileo-caecal region, and not invading the muscular coat; (ii.) occupying the subserous lymphatics and forming a diffuse rigid sheath around the intestine which, from invasion of its muscular coats, becomes dilated. Severe diarrhœa is a prominent feature. The cause of lymphadenoma is still unknown, but since its histology has been more strictly defined it appears probable that Dr. Pitt's second group of cases, at any rate, is not lymphadenoma but lymphosarcoma. Dr. Salaman, indeed, concludes that the intestinal tract is never affected by true lymphadenoma, but this view would seem to be extreme. Enlargement of Peyer's patches does occur in cases of lymphadenoma elsewhere in the body, but there are no diagnostic symptoms whereby this can be recognised during life.

PRIMARY MALIGNANT DISEASE OF THE SMALL INTESTINE is rare, and contrasts with the common occurrence of carcinoma in the large intestine. By combining a number of published statistics, Brill found that of 3563 cases of intestinal cancer (probably including sarcoma as well as carcinoma) 89 or 2·5 per cent were in the small intestine. Malignant disease of the small intestine may be carcinoma or sarcoma. Carcinoma is more often found in the duodenum. I have collected 54 cases of carcinoma primary in the duodenum, and 19 in the jejunum and ileum. In some statistics the ileum has been credited with a large number of cases, but this is probably because primary carcinoma of the ileo-caecal valve has been counted as arising in the ileum, as in the cases described by Du Castel. The incidence of primary sarcoma of the small intestine is just the reverse; thus, Libman was only able to collect 15 cases primary in the duodenum, while Messrs. Corner and Fairbank tabulated 7 primary in the jejunum and ileum. Primary malignant disease of the small intestine may be conveniently considered as it affects (i.) the duodenum, (*a*) carcinoma, (*b*) sarcoma; and (ii.) the jejunum and ileum, (*a*) carcinoma and (*b*) sarcoma.

Primary Carcinoma of the Duodenum.—It is generally held that carcinoma of the pylorus does not extend into the duodenum, and certainly the cases are very few in which it spreads widely into it; but it must be borne in mind that when carcinoma attacks the pyloric region the situation of the pyloric valve may be difficult to define accurately, and the tumour and the pylorus may be regarded as coextensive. In 10 specimens in which the pylorus had been excised for carcinoma, Cunéo and Lecène found that the growth had extended into the duodenum, as judged by the naked eye, in one instance only, but that microscopically the lymphatics in the duodenum were infected in three others for a short distance, never more than $\frac{1}{4}$ths of an inch.

Primary malignant disease of the duodenum must be distinguished from primary malignant disease of the head of the pancreas, and from

primary carcinoma of the ampulla of Vater. The pathological and clinical features of malignant disease in these two situations differ somewhat from those of primary duodenal carcinoma. Primary carcinoma of the head of the pancreas (*vide* Vol. IV. Part I.) is practically always a spheroidal-celled growth with a large amount of fibrous tissue.

Primary Carcinoma of the Ampulla of Vater, or the cavity inside the biliary papilla into which the terminations of the common bile-duct and Wirsung's duct of the pancreas open, is often confused with primary carcinoma of the duodenal surface of the biliary papilla on the one hand and with primary carcinoma of the termination of the common bile-duct on the other. The parts concerned are so small and in such close relation to each other that error is not unnatural. Primary carcinoma of the lining membrane of the ampulla of Vater is rare. I have collected 19 cases. It is a small growth, columnar-celled histologically, produces absolute and progressive jaundice, and is clinically indistinguishable from primary carcinoma of the lower end of the common bile-duct.

Morbid Anatomy.—Primary carcinoma may be limited to any one of the three parts of the duodenum, or may extend from one part to another; usually the growth is comparatively small and produces an annular stricture. A case of two distinct carcinomatous growths, one in the first part and the other, regarded as due to implantation from the first, in the second portion of the duodenum, has been recorded (Pye-Smith). In 51 collected cases the first part of the duodenum was affected alone in eight, and together with the second part in seven more. The second part was the site of a primary carcinoma confined to this portion in 29; in seven others, as already stated, the first part was also affected, and in one a growth had extended from the third portion. The third portion was the starting-point in 7 cases, in one of which it had spread up into the second portion. The large proportion of cases arising in the second part depends on the comparative frequency with which primary carcinoma attacks the mucous membrane of the biliary papilla. As already pointed out this is quite distinct from primary carcinoma of the ampulla of Vater.

Primary carcinoma of the duodenum is usually a columnar-celled growth, in rare cases it is spheroidal-celled. It may undergo colloid degeneration (Letulle). The columnar-celled form originates in the surface mucous membrane; it is possible that the spheroidal-celled growths are derived from Brunner's glands or from outlying pieces of the pancreas embedded in the walls of the duodenum. In about 10 cases (Perry and Shaw 5, Nattan-Larrier 5), carcinoma has arisen in the site of a pre-existing ulcer; but ulcer cannot be an important factor in the causation of duodenal carcinoma, because ulcer is almost confined to the first part, while carcinoma is much more frequent in the second. We may surmise that the reason why the biliary papilla is a favourite site for carcinoma is on account of some foetal displacement or irregular inclusion of epithelial cells during the outgrowth of the hepatic diverticula from the primitive duodenum.

Primary carcinoma of the duodenum is much commoner in males than

in females; in 54 cases 41 were males and 13 women. It occurs at about the same age as primary carcinoma of the stomach and large intestine; in 53 cases the average age was 51·6 years, 50·8 in 13 women, 51·8 in 40 men. The extremes were 80 (in a man) and 24 years (in a woman).

Symptoms.—The symptoms common to carcinoma, in whatever part of the duodenum it be situated, have a general resemblance to those of pyloric obstruction. There is progressive loss of weight and strength due to starvation, with diminished output of chlorides in the urine and increased indicanuria. The abdomen is flat in the lower part, but there is progressive dilatation of the stomach, with visible peristaltic contractions; from time to time copious vomiting of fluid food occurs, after which the patient feels better, and there may be scanty haematemesis. The vomiting gives rise to thirst, scanty urine, and constipation. Occasionally, however, there is diarrhoea, which may alternate with constipation. In some instances there is melaena. A tumour and transmitted aortic pulsation may be palpable in a few instances. There is usually pain in the right hypochondrium, which may extend to the epigastrium, or spread more widely over the abdomen. The symptoms special to carcinoma in the different parts of the duodenum will now be described according as the growth is above the biliary papilla (juxta-pyloric, supra-ampullary, or roughly the first part), in the neighbourhood of the biliary papilla (peri-vaterian, circum-ampullary, or roughly the second part), or below the biliary papilla (juxta-jejunal, infra-ampullary, or roughly the third part of the duodenum).

In *supra-ampullary* carcinoma, which is either in the first part or in that portion of the second stage of the duodenum above the biliary papilla, the symptoms very closely resemble those of pyloric cancer. Dilatation of the stomach and vomiting of material devoid of bile occur, but free hydrochloric acid may be present (Czygan), and there is seldom any blood. The motions contain bile, and there is no jaundice unless there be some complication, such as compression of bile-ducts by a secondary growth. A tumour is palpable in 60 per cent of the cases (the Fenwicks), and is tender and less movable than a carcinomatous pylorus.

Circum-ampullary Carcinoma.—Primary carcinoma of the second part of the duodenum most commonly starts in the mucous membrane covering the biliary papilla, but it may arise elsewhere, and need not invade the papilla; the two conditions give rise to entirely different symptoms, referable to obstruction of the common bile-duct or to stenosis of the duodenum respectively. Carcinoma of the duodenal surface of the biliary papilla is characterised by obstructive jaundice, with distension of the gall-bladder at a very early date when the growth is quite small; there may also be diarrhoea from irritation set up by the growth; as the result of ulceration or sloughing of the growth the biliary obstruction may be relieved and the jaundice may disappear. Infection may then ascend into the duct and give rise to suppurative cholangitis, fever, rigors, and

death. Primary carcinoma of the duodenal surface of the biliary papilla must not be confused with carcinoma of the ampulla of Vater or with primary carcinoma of the head of the pancreas, in both of which the jaundice is progressive, does not intermit, and is seldom complicated by suppurative cholangitis. When the growth arises in the second part of the duodenum but not in the biliary papilla the symptoms vary according to its position above or below the entrance of the bile-duct; if above, the symptoms are those of supra-ampullary carcinoma; if below, those of infra-ampullary carcinoma. The distinction mainly rests on the absence or presence of bile in the vomit. Carcinoma starting at a distance from the biliary papilla may spread to it and give rise to the symptoms described above.

When the growth is in the third or in the portion of the second stage of the duodenum below the biliary papilla, or *infra-papillary*, the bile and pancreatic juice regurgitate through the dilated duodenum into the stomach; and their presence in the vomit should arouse suspicion that the case is not one of pyloric obstruction or of cancer in the first part of the duodenum. When bile is constantly present in the vomit, the ferments of the pancreas, especially the fat-transforming steapsin, may be searched for as a confirmatory sign of regurgitation through the pylorus. The presence of alkaline bile and pancreatic juice in the stomach neutralises the hydrochloric acid of the gastric juice, which is therefore usually, though not always, found combined and not free. The reflux of the bile into the stomach accounts for its diminution or absence in the faeces. If a tumour is felt it is fixed, and thus resembles that of pancreatic cancer, and differs from the movable tumour of carcinoma of the pylorus. The duodenum is more extensively dilated than in supra-papillary carcinoma, and the accumulation of food in both cases may lead to the regurgitation filling up a stomach which has been emptied over-night.

In cases in which the duodenum is not narrowed, the backward pressure and dilatation of the duodenum and stomach need not be present; though vomiting may be severe, and the aspect of the case will perhaps be rather that of gastric ulcer.

The course of the disease thus has a very general resemblance to carcinoma of the stomach. Death usually results from asthenia and starvation; but in a few cases has been from loss of blood due to ulceration of the pancreatico-duodenal arteries by the growth. According to Pic, the duration of the disease is from three months to a year.

Diagnosis.—A passing reference has already been made to the diagnosis of carcinoma of the duodenum from malignant disease in the stomach, pancreas, and adjacent parts.

Malignant disease of the first part is practically indistinguishable from pyloric carcinoma; and, like it, has to be diagnosed from gastric ulcer, simple dilatation of the stomach, cicatricial contraction of an ulcer near the pylorus, compression of the pylorus or of the first part of the duodenum by tumours or by inflammatory adhesions due to cholecystitis. Duodenal carcinoma, it is true, runs a more rapid course than many

cases of carcinoma of the pylorus, but this factor would not be of much use in diagnosis.

The absence of localised tenderness, the scanty haematemesis, the relation of pain to the ingestion of food, and the presence of a tumour would distinguish it from gastric ulcer. The presence of a tumour and the cachexia would separate it from simple dilatation of the stomach. The history of cholelithiasis and the slow progress of the case should point to peritoneal adhesions around the pylorus; while the special characters of an abdominal aneurysm, a cancerous or calculous gall-bladder, or a hydronephrosis, would distinguish them from carcinoma of the first part of the duodenum.

When obstruction occurs lower down in the small intestine or in the colon, and gives rise to obstruction, the abdomen will become more generally distended than in duodenal stenosis, in which the stomach is dilated but not the small intestines.

Treatment is the same as that of gastric carcinoma; the medical treatment by appropriate diet by the mouth, nutrient enemas, washing out the stomach, and, if necessary, the relief of pain by hypodermic injections of morphia.

Surgically, excision of a growth in the third part of the duodenum has been successfully performed (Syme). Otherwise the prognosis is necessary fatal. Gastro-jejunostomy is of course a palliative only, but should be adopted if the vomiting be urgent, as so often it is, and if the patient be in a fairly fit state to bear the shock of the operation.

Primary sarcoma of the duodenum is very rare; in 1900 Libman collected 15 cases only. The growth usually occupies a considerable extent of the duodenum, and often occurs in plaques resembling hardened Peyer's patches. A lymphosarcoma of the stomach may spread by continuity along the walls of the duodenum so that a considerable extent of the alimentary canal is implicated. Infiltration of the wall of the duodenum so weakens its resistance that dilatation results, and there is of course no obstruction as in carcinoma. The growth is usually a round-celled or a lymphosarcoma, and may lead to considerable ulceration. Repeated or fatal haemorrhage may thus result. In a case with repeated attacks of melaena simulating duodenal ulcer, which I saw on several occasions, there was a pedunculated sarcoma; in another case with widespread ulceration of the growth implicating the biliary papilla but not producing jaundice, there were eructations like rotten eggs, rapid emaciation, and fatal haemorrhage from erosion of the inferior pancreatico-duodenal artery; gastric ulcer had been diagnosed.

Primary malignant disease of the jejunum and ileum is rare, carcinoma being even less frequent than sarcoma.

Primary Carcinoma is extremely rare; thus, in 41,838 autopsies at Vienna during the years 1870-93 there were 10 cases only, all in the ileum (Nothnagel); in more than 2200 autopsies at the Johns Hopkins Hospital there were 2 cases, both in the upper part of the ileum. Lubarsch in 1888 collected 35 cases of primary carcinoma of the ileum.

As already noted, carcinoma of the ileo-caecal valve, which is by no means rare, must not be confused with primary carcinoma of the ileum. In 19 cases of primary carcinoma of the jejunum and ileum that I have tabulated, the average age was forty-six years, being 50·2 years in the 11 males and 40 years in the 8 females. Histologically, the growth may be either a spheroidal-celled (11 cases), or a cylindrical-celled carcinoma (6 cases), like that in the colon.

A distinct group of cases of multiple primary carcinoma has been isolated by Bunting, to whose collection of 7 cases one recorded by Tanberg may be added; histologically the growths were spheroidal-celled and shewed vacuolation, closely resembling spheroidal-celled carcinoma of the vermiform appendix. The tumours were small, did not give rise to any clinical manifestations, and except in one case (Ransom) there were no secondary growths. In their benign character they further resemble the similar condition in the appendix. The multiplicity of the tumours is not due to implantation or to metastasis, but to independent foci of growth.

Primary carcinoma may produce an annular stricture of the small intestine, but from the fluid nature of the contents does not give rise to symptoms of obstruction so early as does carcinoma of the colon. A palpable tumour is not so frequently present as in sarcoma. The growth may sink down into the lower part of the abdomen, especially the right iliac fossa, and be very freely movable; it may, however, become adherent to other viscera, such as the colon, and give rise to a fistulous communication. The usual symptoms are attacks of colic, nausea, vomiting, and constipation. For the diagnosis from carcinoma of the colon see p. 758. Treatment by excision has given good results.

Primary sarcoma of the jejunum and ileum is commoner than carcinoma. Messrs. Corner and Fairbank have collected 19 cases primary in the jejunum, and 28 in the ileum. It is about three times commoner in men than in women; it may occur at any age, but is commoner before the age of forty, and a considerable number have been in quite young subjects.

Sarcoma of the small intestine usually starts in the submucous coat, but a few examples of myosarcoma have been reported. The growth may be pedunculated, form a flat growth, or infiltrate the wall of the intestine diffusely. Secondary growths occur in about one-third of the cases. The tumour is generally a round-celled sarcoma, and invades the muscular coats. It may narrow the lumen either by thickening of the coats or by the projection of masses of growth into the lumen, but the peculiar point about primary intestinal sarcoma is that in some cases (9 out of 65, Corner and Fairbank) the bowel is dilated. The growth is usually of a considerable size, from that of an orange upwards, and may shew necrosis and even soften down into a pseudo-cyst. Sarcoma of the intestine may be round-, spindle-, or mixed-celled, or in rare cases a myosarcoma. There is still some difference of opinion as to the nature of the intestinal new-growth most commonly called lymphosarcoma, but

described by Dr. Pitt as that form of lymphadenoma (*vide* p. 577) which forms a diffuse sheath for the intestine. It differs from other forms of sarcoma in being more diffuse but less malignant, and in extending almost entirely by continuity or by contact. Libman has suggested that it may be infective in origin.

The *symptoms* of sarcoma of the small intestine are generally not, like carcinoma of the part, those of chronic obstruction, but are somewhat vague in character:—pain, diarrhœa, anæmia, and loss of flesh, associated with the presence of a movable tumour which, as time advances, tends to gravitate towards the lower part of the abdomen; appendicitis may thus be imitated. Irregular fever occasionally occurs, and exceptionally a high temperature has been associated with hæmorrhages and subcutaneous emphysema (Fagge, Morton), presumably from infection with micro-organisms, among which were some with the power of producing gas. Ascites may occur, but depends on secondary growths in the glands or peritoneum, rather than on the primary growth. Some pedunculated tumours arising in the intestine may have the structure of spindle-celled sarcoma. They may give rise to intestinal obstruction and to intussusception. According to Lecène the duration of life from the onset of symptoms is four months in round-celled, and eight to ten months in spindle-celled sarcomas.

The only satisfactory *treatment* is excision of the growth by the surgeon; medical treatment can be but palliative.

Secondary Growths or Invasion of the Small Intestine.—*Secondary malignant disease of the duodenum* is very rare, but new growth in the neighbourhood may extend into it by continuity. Carcinoma of the stomach hardly ever spreads into the duodenum (*vide* p. 577), but lymphosarcoma of the stomach may do so very freely. Carcinoma of the ampulla of Vater may project into the duodenum, and carcinoma of the head of the pancreas may invade and lead to ulceration of the duodenum, though in such cases it may be difficult to determine the starting-point of the growth. Primary carcinoma of the gall-bladder may invade the duodenum and produce stenosis; but when it does give rise to a bimucous fistula, primary carcinoma of the gall-bladder usually opens into the transverse colon, less often into the duodenum; this is rather remarkable, since in ulceration of a gall-stone out of the gall-bladder into the alimentary canal the duodenum is much more often implicated than the colon. I have seen a soft round-celled sarcoma arising from the retroperitoneal space surround the first part of the duodenum, and give rise to the symptoms of pyloric obstruction, and in another case a necrotic pseudo-cyst in a retroperitoneal sarcoma opened into the second part of the duodenum.

When there is infection of the peritoneum with malignant disease, the jejunum and ileum may become invaded from without by the secondary growths. In generalised sarcomatosis the mucous membrane may contain numerous small secondary growths which may become

pedunculated; this may occur in melanotic sarcoma in which I have seen tiny growths in the villi of the small intestine. Secondary growths, by invading the muscular coat, may draw on the circumference of the bowel and lead to narrowing or kinking, or may perhaps dispose to invagination; but as a rule secondary growths give rise to no symptoms. The interesting process of implantation from a growth in one part of the intestine to another forms another and probably very rare method of metastasis.

The small intestine may be invaded by continuity of malignant disease originating in other abdominal organs, and may become compressed and the lumen obstructed. When the invading growth starts in the colon or stomach a fistulous communication may be set up. A specimen in St. George's Hospital Museum shews a communication between the jejunum and a primary carcinoma of the lower end of the sigmoid flexure; during life there had been diarrhoea from the short circuit. The small intestine rarely becomes surrounded and invaded in sarcomatous or other growths springing from the retroperitoneal space.

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APPENDICITIS

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Definition.—Pathologically, the name "appendicitis" implies an inflammation of the vermiform appendix; clinically, an inflammation of the vermiform appendix, of the peritoneum in its immediate vicinity, and of neighbouring structures, especially of the caecum, of the end of the ileum, and of the ileo-caecal junction and valve. Inflammations which begin in the vermiform appendix, and spread to structures more distant from it, are usually referred to as complications; for example, inflammation of the colon, of the urinary bladder, of the broad ligament of the uterus, of the Fallopian tube, or of the ovary. Several names have been applied to the group of clinical symptoms set up by inflammation, beginning in the vermiform appendix and spreading to the structures in its neighbourhood. Amongst these, "typhlitis" and "perityphlitis" were amongst the best, but have been supplanted by the name "appendicitis," coined by Fitz, which, although objectionable to the philological purist, serves to indicate an inflammation that begins and is for a variable period confined to the appendix vermiformis. Those interested in the history of these various names should consult the monumental work of Kelly and Hurdon, to whom, together with Deaver and Kelynack, I am greatly indebted.

Some of the names given to diseases of the appendix are ambiguous, and therefore misleading. When "simple" appendicitis is spoken of, does it mean that the clinical symptoms are simple, or the inflammatory process, or the end, which may be abscess, perforation, or gangrene? Some speak of "perforative appendicitis," or of "gangrenous appendicitis," as though there was one kind which ended in perforation, and another which ended in gangrene. Yet an inflamed appendix may at the same moment display both perforation and gangrene (14). The

name "tuberculous appendicitis" is an instance of scientific terminology. It implies the cause of the appendicitis, the presence of the tubercle bacillus, and enables us to name correctly the results, as, for instance, when we speak of tuberculous ulceration. Until, therefore, the etiology of the various kinds of appendicitis is known, it will be safer to speak of appendicitis and qualify the term by adding the most distinctive feature: as, for example, appendicitis with ulceration of the mucosa, and so forth. In most cases the cause of the appendicitis is in the appendix itself. But, on the other hand, appendicitis may be secondary to disease of the intestines, such as colitis or enteric fever; to disease of the peritoneum, such as tuberculous peritonitis; or disease of the uterus, broad ligament, and Fallopian tube.

Clinicians have attributed appendicitis to influenza, tonsillitis, gout, rheumatism, and so forth. As yet these assumptions do not rest upon a scientific basis, but it seems reasonable to suppose that some may act as predisposing causes. It is, however, unnecessary to point out that common diseases might be expected to coincide.

The anatomy of the vermiform appendix has a profound influence upon its diseases. No other organ is so variable, and none when diseased can give rise to such diverse symptoms. Absence of the vermiform appendix is exceedingly rare. Huntington is one of the few who have failed to discover any trace of its presence. But absence is too unusual to serve as an excuse for failure to find an appendix. Surgeons who have not found the appendix probably overlooked one which was hidden in the ileo-caecal or retro-colic pouch; or one which was exceedingly thin, and therefore easily missed amidst vascular adhesions. Obviously, the diagnosis of appendicitis implies the presence of an appendix.

M'Burney's name is now inseparably connected with the topographical anatomy of the vermiform appendix. His statement is as follows: "I believe that in every case the seat of the greatest pain, determined by the pressure of one finger, has been exactly between an inch and a half and two inches from the anterior spinous process of the ilium in a straight line drawn from that process to the umbilicus. This point indicates the situation of the base of the appendix where it arises from the caecum, but does not by any means demonstrate, as one might conclude, that the chief point of disease is there" (16). This rule defines with sufficient precision the locality of the base of the vermiform appendix. In 200 operation cases, the appendix was in the iliac fossa in 140; in the pelvis in 37; in the retro-colic and ileo-caecal fossae in 17 (11 and 6 respectively); external to the caecum or right colon in 3; and in hernial sacs in 2 cases. These proportions are the same as those derived from anatomical examinations. These variations in position are a fruitful cause of difficulties and errors in diagnosis. In females, an inflamed appendix when in the pelvis may be mistaken for ovaritis, salpingitis, haematosalpinx, and so forth. On many occasions an inflamed, gangrenous, or perforated appendix in the pelvis has been overlooked, with disastrous results. An empyema of a retro-colic appendix has

been mistaken for a tuberculous kidney, and a chronic retro-colic appendicular abscess for malignant disease of the colon. The part of M'Burney's work which refers to the seat of pain in appendicitis will be referred to hereafter. Some of the unusual positions of the vermiform appendix are explained by the course which the caecum pursues during the development of the colon. At about the third or fourth month of intra-uterine life, the caecum and appendix are situated near the centre of the abdomen. As the colon grows, they pass round towards the right beneath the liver, and descend thence into the right iliac fossa. So then, as in a specimen in the museum of St. Bartholomew's Hospital, the caecum and appendix may both remain high up beneath the liver, or be arrested in any part of their descent towards the right flank. A patient with septic peritonitis had such an undescended caecum with the appendix at its outer side on a level with the iliac crest. Or the caecum and appendix may continue their descent beyond the iliac fossa and lodge in the pelvis. With this kind of pelvic appendix, the caecum is always very capacious. During its passage round the right side of the abdomen into the iliac fossa, the right colon has a mesentery common to it and the small intestine. When this mesentery is retained, the caecum and appendix have an extensive range of movement within the abdomen. The name "floating caecum" is given to this condition. It conduces to intussusception, volvulus of the caecum, and torsion of the vermiform appendix. It is during the descent of the caecum into the iliac fossa, and during the disappearance of the original mesentery of the colon, that the retro-colic and ileo-caecal fossae are formed. It is also during the descent that the vermiform appendix becomes immured within them.

Ordinarily, the appendix is about three and a half inches long, but from being a mere tag it may attain a great length. An appendix twelve and seven-eighths of an inch long probably heads the record (Grauer). From its origin (from the caecum in the iliac fossa), a very long appendix may adhere to the pelvic viscera, to the bottom of Douglas's pouch; or, wandering upwards, a long appendix has adhered to the mesentery, to the transverse mesocolon, to the liver, or to the gall-bladder. A great many instances are recorded of strangulation of intestines beneath the adherent appendix. The search for a very short appendix arising from the outer side of a partially descended caecum has occupied two hours. In infancy and childhood the appendix is relatively larger than in the adult. The diameter of the vermiform appendix is about one-third of an inch, and is nearly the same throughout. The lumen occupies about one-third of the diameter. But great variations in the thickness of the coats and in the size of the lumen are met with. An appendix not much more than one line in thickness has been removed, and would perhaps not have been found if adhesions had existed. The normal appendix is of a yellowish-pink colour, and its encircling vessels are hardly visible to the naked eye. Vessels which can easily be seen indicate inflammation. The sense of touch distinguishes the small, round, firm appendix from other abdominal contents, but not

easily from the Fallopian tube or from the ureter. Oftentimes the appendix contracts when touched, and becomes shorter, thicker, and harder. Faecal concretions can be felt inside the appendix, and are an indication for its removal. When the appendix is distended with faecal contents, they may often be seen through its walls thinned by stretching. The way in which the appendix begins may make it more liable to become the seat of faecal accumulation or of foreign bodies. In the foetal type of Sir Frederick Treves, the caecum has a funnel-shaped entrance into the lumen of the appendix. This was met with 13 times out of 54 consecutive operation cases. One of the 13 had undergone fibrous obliteration of the lumen, 1 contained a pin, and 11 were full of faeces or faeces with concretions. The combination of foetal type of appendix with the pelvic position, which is by no means infrequent, increases the liability to faecal accumulation. But ordinarily the appendix begins at the back and inner side of the caecum, about an inch from the ileo-caecal valve. The opening is usually oblique, like the opening of the ureter into the bladder, so that the mucous membrane is prolonged towards the right from the acute angle of junction of the caecum and appendix. This prolongation is the valve of Gerlach. It is very doubtful whether it can keep faeces or foreign bodies out of the appendix; but it seems reasonable to suppose that their passage is not so easy through the oblique opening as through the wide, funnel-shaped mouth of the foetal type. The appendix may originate from any part of the caecum. One which begins at the back, and is hidden away in the retro-colic or in the ileo-caecal fossa, may be difficult to find, or, indeed, may not be found by one who is not acquainted with this arrangement.

The coats of the vermiform appendix are (1) the peritoneal, (2) the muscular, (3) the submucous, and (4) the mucous. The peritoneal and muscular coats, especially the circular muscular coat, form a rather unyielding ring encircling the loose and voluminous mucous membrane. When the mucous and submucous coats are acutely inflamed great oedema and swelling result. In this way the lumen is blocked, and the muscular and peritoneal coats distended and stretched. Distension and stretching of the peritoneal and muscular coats are among the chief causes of appendicular pain. When the appendix is perforated, the mucous lining, owing to its looseness, protrudes through the opening and may prevent it from healing (*vide* Fig. 24). In this way an intra- or extra-abdominal appendicular fistula is formed. The peritoneal coat is so thin that it can hardly be seen with the naked eye in transverse sections such as are used for the microscope. Although only half or one millimetre thick, it is tough and resistant, and the last to give way when the appendix is the seat of concretion, abscess, or ulceration. Its surface is covered with a single layer of very thin endothelial plates; beneath these is a small quantity of loose, delicate connective tissue, which contains the small subperitoneal arteries, veins, nerves, and lymphatics. This connective tissue is continuous with that amongst the

longitudinal and circular muscular coats, and with the connective tissue of the submucous and mucous coats at each hiatus muscularis. The latter are gaps which the author discovered in the muscular walls of the appendix, through which not only the connective tissues of the peritoneum and of the mucous coat are continuous, but also the lymphatics, blood-vessels, and nerves. This explains why inflammation of the mucous membrane spreads so rapidly to the peritoneum—a point duly recognised in the name "perityphlitis," once applied to appendicitis, which aptly designates this most important peculiarity.

The longitudinal muscular coat, lying beneath the peritoneal, is a continuation of the three longitudinal bands of the large intestine. Unless hidden by inflammation or adhesion, the anterior longitudinal muscular band of the colon and caecum is a guide to the base of the appendix. The longitudinal coat is half or one millimetre thick. Its fibres are thicker at some parts of the circumference than at others, and this is supposed to be a trace of the three bands of the large intestine. When the appendix is distended its longitudinal muscular fibres go apart, and are little, if any, protection against perforation. When contracted, they may shorten the appendix by a quarter of its length. The circular muscular coat is usually about one millimetre thick. When the appendix is distended it becomes thinner, but is still a barrier against perforation. At the attachment of the meso-appendix it has gaps (hiatus musculares) in it one millimetre wide and easily to be seen with the naked eye. These transmit vessels, nerves, and lymphatics, and are filled with connective tissue. They have already been mentioned as places where there is direct continuity between the arteries, veins, lymphatics, nerves, and connective tissues of the peritoneal and mucous coats.

The submucous is separated from the mucous coat by the thin muscularis mucosae. But as these muscle-cells cannot always be seen, the line of the separation is an artificial one. The submucous connective tissue is loose, and has in it capacious lymph-spaces, lymphatics, and veins. It may have a few fat-cells in it. It easily becomes swollen and oedematous, helping to lessen the lumen. In some kinds of appendicitis, it is the seat of fibrous hyperplasia leading to constrictions, or even to complete obliteration of the lumen. When the lumen is distended with mucus, pus, or faeces, the submucosa is compressed against the circular muscular coat, and looks like a thin, fibrous layer.

The mucosa is about ·75 mm. thick, rather loose and voluminous, and in longitudinal folds, which project into the lumen. When the lumen is distended these folds disappear. As a result of its looseness, the mucosa protrudes through perforations, and may prevent their closure and cause a permanent fistula (*vide* Fig. 24). Like the mucous membrane of other parts of the intestine, that of the appendix is tough and inextensible. It may be inferred that after its folds have been obliterated by distension it soon gives way. Next to the lumen the mucosa has a single layer of columnar epithelial cells upon a basement membrane, and its tubular glands

have a similar lining. This epithelial layer is the protective barrier against the passage of bacteria and toxins into the venous and lymphatic systems. Its integrity is of the utmost importance, its destruction being the starting-point of some of the most deadly forms of appendicitis. The tubular glands of the mucosa are about half a millimetre long, and are embedded in the delicate adenoid or lymphoid tissues of the mucosa. Usually they are like a test-tube, but the blind end is sometimes bifid, and now and then trifid. When the lumen is distended, the tubular glands widen out into shallow bays, and may at last become obliterated. Faecal and septic material may be forced into their lumens, and besides

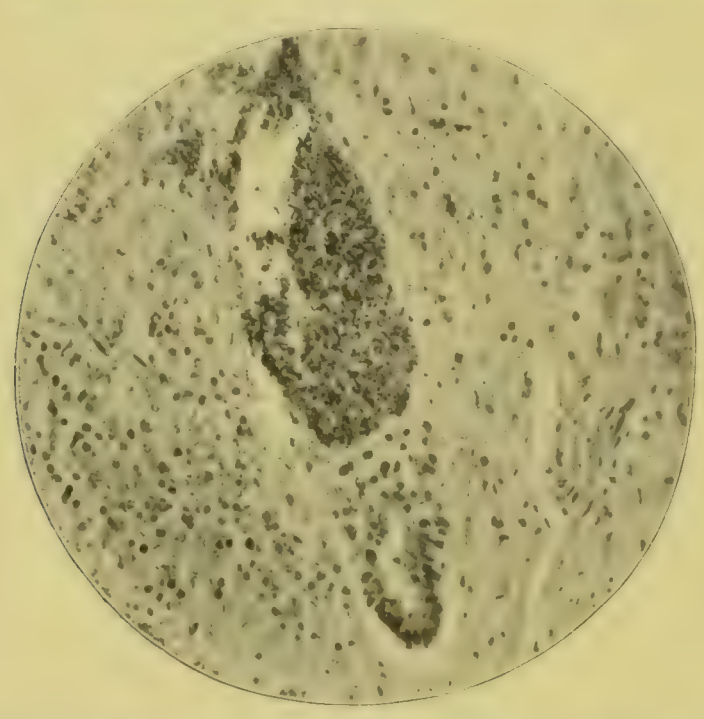


Fig. 19.—Bacteria invading mucosa along the track of a tubular gland. The lumen is above. ×200. Penetration of bacteria by way of the tubular glands is quite common in cases in which the lumen is full of septic contents.

distending them destroys their epithelial lining (Fig. 19). In this way bacterial, purulent, and even faecal matter makes its way into the lymphatics of the mucosa and submucosa. At all stages of its existence the mucosa contains numerous lymphoid follicles. These are globular, ovoid, or pyriform bodies about a millimetre in diameter, and easily visible to the naked eye. An appendix of average length probably contains from one hundred and fifty to two hundred follicles, but Kelly and Hurdon give double this estimate. Each follicle, as in other regions, consists of a cortical part, which is crowded with concentrically arranged lymphoid cells and stains deeply, and of a central germinal area which has larger lymph-canaliculi and contains fewer cells. Towards the lumen the lymphoid follicles are covered with the single layer of columnar epithelium, whilst towards the submucosa they are,

as it were, capped with a wide semilunar lymph-space (the basilar lymphatic), which opens freely into the lymphatics of the submucosa and onwards into the lymphatic system of the peritoneum and mesentery. When the layer of epithelium upon the surface of the lymphoid follicles is destroyed by ulceration—a common event—bacteria crowd into the tissues of the follicles and into their basilar lymphatics (Fig. 20). Thus lymphadenitis and lymphangitis play a prominent part in the morbid anatomy and pathology of appendicitis.

The follicles, when inflamed, may swell enormously; the lymphatics may be the seat of acute and widespread septic inflammation; and the lymphatic glands too may be greatly swollen and inflamed. A proper

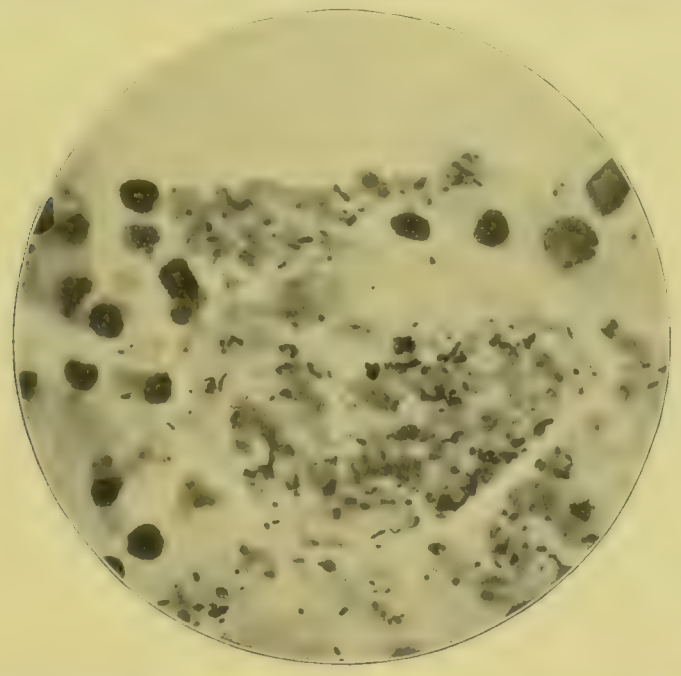


FIG. 20.—Part of the wall of an inflamed appendix from which the lining epithelium and tubular glands have disappeared. A lymphoid follicle is seen crowded with bacteria, which are also seen in its basilar lymphatics. The lumen is above. ×1000.

knowledge of the origin and distribution of the appendicular lymphatics is essential. The lymphatics begin in lymph-canaliculi and minute channels beneath the epithelium of the lumen and of the tubular glands, so that destruction of the epithelium lays them open to the infective contents of the lumen. Thence they pass into the basilar lymph-spaces and into the plexus about the base of the tubular glands, and onwards into the larger lymphatics of the submucosa. The latter gain the outside by passing through the gaps in the muscular coats (hiatus musculares), and thus inflammation within the appendix quickly spreads to its peritoneal covering and to the meso-appendix. After leaving the appendix, the lymphatics accompany the appendicular blood-vessels in the meso-appendix, and in great part pass, with the posterior ileo-caecal artery, behind the end of the ileum. Here they sometimes enter a small group

of lymphatic glands and pass onwards to the lymphatic glands of the mesentery, and to those along the inner side of the right colon. When the latter are inflamed the pain and tenderness of appendicitis may extend into the region of the kidney or of the gall-bladder, and thus an element of confusion is introduced into the diagnosis. There is occasionally a lymphatic gland (Clado's gland (6)) in the meso-appendix close to the ileo-caecal junction; others lie behind the end of the ileum, but the main group is situated at the end of the mesentery in the ileo-colic angle. In tuberculosis and carcinoma of the appendix these various glands enlarge. I have excised the group at the ileo-colic angle in carcinoma of the appendix and caecum. Glands behind the lower end of the right colon also become enlarged in appendicular diseases. The lymphatic system has been studied by anatomists with the aid of injections, and some have assumed that the flow of injections is a guide to the direction pursued by disease. But cancer, for instance, does not by any means follow the direction of the lymph-stream, and is not resisted by the presence of valves. Septic inflammations, too, do not obey the laws laid down by anatomists. The clinical study of appendicitis and observations made during operations shew that after leaving the meso-appendix the lymph flows in directions not revealed by injections. It is quite common to feel the glands enlarged along the course of the external and common iliac vessels, and also those within the pelvis, about the internal iliac vessels. The presence of the latter affords an explanation of a very common clinical sign of appendicitis, namely, tenderness on pressure at the right side of the pelvis by the finger pushed high up the rectum. In addition, there are lymphatic channels between the vermiform appendix and the right broad ligament of the uterus. So that I have seen the right broad ligament inflamed and swollen in appendicitis, and septic endometritis spread to the broad ligament and onwards to the vermiform appendix. As Clado has shewn, a knowledge of the anatomy and development of the structure which I named the plica vascularis affords an explanation of this relationship of lymphatics (15). Lymphatics also pass across behind the pubic bones from the right side of the pelvis to the left. So that with an acute appendicular abscess behind the right arch, the lymphatic glands along the left external iliac vessels may become inflamed and threaten to suppurate. It is probable that similar intercommunications take place behind Douglas's pouch. A female with appendicitis had an inflamed gland near the left common iliac artery.

The success of appendicectomy depends in no small degree upon the avoidance of haemorrhage during and after the operation. When the position of the vessels is known they can be secured before they are cut, and none overlooked. The anatomy of the vessels also has an influence upon the pathology and clinical symptoms of appendicitis. It is the most peripheral part of distribution of the superior mesenteric artery, and a likely spot, therefore, for the arrest of any organisms which may be circulating in the blood. The main arterial supply of the vermiform appendix is derived from the posterior ileo-caecal artery, and it may

receive small branches from the anterior ileo-caecal. The posterior ileo-caecal artery passes down from its parent trunk, the ileo-colic, behind the end of the ileum, and, after giving off branches to the right colon, to the end of the ileum, and to the lower segment of the ileo-caecal valve, ends in caecal and appendicular branches. Owing to identity of blood-supply, the ends of the ileum, the caecum, the right colon, and the ileo-caecal valve participate in inflammation of the vermiform appendix. We have here a clue to one of the causes of the constipation, flatulent distension, and colic so commonly met with in appendicitis. The anterior ileo-caecal artery also supplies the ileum, the right colon, caecum, and the front segment of the ileo-caecal valve, but it does not always reach as far as the appendix. In the ileo-caecal valve it has a free anastomosis with the posterior ileo-caecal artery. The posterior ileo-caecal artery supplies the appendix with two sets of branches. Its caecal branches give off an artery at the base of the appendix. This branch runs towards the tip in the junction of the appendix and meso-appendix, and anastomoses with the meso-appendicular arteries. This small vessel, being close to the caecum, is apt to be missed by the ligature placed upon the meso-appendix, and requires, therefore, an additional suture. Occasionally the caecal branch of the ileo-caecal artery sends a small twig along the appendix at the side farthest from the meso-appendix. This little artery may also require a separate suture. In embryonic life, before the meso-appendix and its vessels appear, these caecal arteries are the only blood-supply the appendix has. Occasionally this embryonic condition persists, and the appendix has no mesentery or meso-appendicular artery. In these circumstances its blood-supply must be rather exposed to obstruction. The main artery from the posterior ileo-caecal runs behind the end of the ileum into the free edge of the meso-appendix. It usually reaches the appendix at about the junction of its middle and distal third, and, rapidly diminishing, runs onwards towards the tip. On its course through the meso-appendix it supplies three or four branches to the proximal two-thirds of the appendix, but long appendices may have a greater number of branches. As in other parts of the intestinal tract, the arteries do not anastomose with any degree of freedom. After reaching the appendix, the arteries, as in no other parts of the alimentary track, divide into an external set of branches which ramify in the peritoneum and supply it and the muscular coats; and into an internal set, which enter at the muscular hiatuses and supply the muscular coat, the submucosa, and the mucosa. From the submucous vessels the lymphoid follicles are supplied with blood. A series of capillaries run inwards between the tubular glands, and, after forming loops beneath the epithelial lining of the lumen, run back again to empty their blood into the veins of the submucosa. The veins of the vermiform appendix correspond with the arteries and form a submucous and peritoneal plexus, and in the meso-appendix run with the arteries. They are very capacious throughout, with thin and distensible walls and no valves. These capacious vessels run a straight and direct course through the meso-

appendix into the ileo-colic and into the superior mesenteric veins, and thence their blood passes onwards into the portal trunk. In appendicitis, infective clots sometimes form in the veins of the appendix and of the meso-appendix. In a recent case the clotting had extended into the veins of a loop of ileum which was nine inches long and ended about six inches from the ileo-caecal junction. The patient recovered. In another case of appendicitis all the superior mesenteric veins were thrombosed; portal thrombosis also supervened. Bacteria, or bacteria carried in clots, have an open way along the capacious, valveless veins from the appendix to the liver, and hence hepatic suppuration is a not infrequent complication. The thin-walled appendicular veins are completely obstructed in torsion of the appendix. The result is the same as in torsion of the kidney, testicle, or ovary, namely, great engorgement, swelling, extravasation of blood, and ultimately gangrene. By means of the right colic veins the appendicular are brought into communication with the systemic system. This intercommunication has by some been thought to explain the abscesses of the lungs and pleurae which sometimes complicate appendicitis. But there is no difficulty in believing that bacteria, which are one-tenth the diameter of red blood-corpuscles, can easily pass through the capillaries of the liver.

The nervous supply of the appendix resembles that of the rest of the intestines. The ganglion-cells of Meissner's plexus are seen at the base of the tubular glands, and those of Auerbach's plexus between the muscular coats. The afferent and efferent nerves pass to and from the solar plexus through the superior mesenteric. In the solar plexus connexions are established with the sympathetic nervous system, with the cerebrospinal system through the splanchnics, and with the right vagus. Morphologically, the vermiform appendix is a median splanchnic organ, and has therefore a bilateral nerve-supply. Through these connexions the pain caused by inflammation, swelling, and distension of the appendix is carried to the sensorium. Also the pains are often referred to the region of the umbilicus, where the somatic divisions of the splanchnic nerves are distributed. This early umbilical pain has often caused the inflamed appendix to be overlooked, especially when occupying the pelvic position. The pain, too, like the nerve-supply, is bilateral.

In transverse sections the lumen of the appendix is irregular owing to the pleats or folds of the loose mucous membrane which lines it. But its lumen may be circular, or trifoliate, or H- or T-shaped, or a narrow slit. The size of the lumen is proportionate to that of the appendix. It is capacious in the long and wide, and narrow in the short and thin. Its calibre is about the same from end to end. Its diameter is usually one-third of that of the appendix. In health it contains a little mucus with shed columnar epithelium, or small quantities of faeces, the presence of which is temporary. A small, thin appendix often has a narrow, irregular lumen, and its distal end may possess none.

The blood-vessels, nerves, and lymphatics of the appendix run in a small mesentery, which also contains some unstriped muscle-fibres and

nat. Occasionally a lymphatic gland is met with near the ileo-caecal angle. In about three cases out of four the attachment of the meso-appendix is continued along the mesentery, just below the last two inches of the ileum. Here the appendicular blood-vessels, nerves, and lymphatics enter and leave. Below, about an inch or an inch and a half from the ileum, it embraces the appendix. To the right it is continuous with the caecum, and to the left its crescentic free edge contains the appendicular artery and veins. The meso-appendix may reach the tip of the appendix, but usually it begins to end at the junction of its middle and distal third. This ordinary type of meso-appendix is very easy to transfix and ligature. It has been mistaken for an adhesion, and torn across, but its anatomy is so characteristic that such blunders should not occur. From a surgical and pathological point of view, the most important variation of the meso-appendix is that in which it is so short that the appendix is bound down in the iliac fossa. In these circumstances the abdominal incision may have to be enlarged, and the appendicular vessels are more difficult to secure. Such an arrangement also enables appendicular inflammation to spread into the retroperitoneal cellular tissues. Now and then the meso-appendix is attached to the caecum. In tying such an one, care is needed lest the gut be nipped in the ligature. The meso-appendix has other variations which are not of importance.

The vermiform appendix may be wholly or partly within either the ileo-caecal or the retro-colic pouch (2). When the mouth of the pouch is closed or hidden, the result is very puzzling. The frequency, too, with which inflamed appendices are found to be hidden away in one or other of these pouches suggests that the position is an unfavourable one. The mouth of the ileo-caecal pouch is seen at the ileo-caecal angle when the caecum and ileum are lifted up. It runs upwards, behind the ileo-colic junction, parallel to the right colon. The small intestine may also enter it to form one of the kinds of retroperitoneal hernia. The mouth of the retro-colic fossa is seen when the caecum is lifted up, and hence it is often called the subcaecal fossa. It runs upwards behind the right colon, and may end in front of the right kidney. When the vermiform appendix is herniated into this fossa, it may give rise to a retro-colic abscess, or even a tumour, which is difficult to tell from a tuberculous kidney or from a malignant tumour of the colon. An abscess at the distal end of a retro-colic appendix was thought to be a pyonephrosis, and the appendix itself to be a ureter. When the appendix is in the retro-colic or ileo-caecal pouch, the tenderness is often absent, being masked by air or faeces in the caecum and colon. In stout patients it is most dangerous to infer much from the absence of tenderness in front of the abdomen, or per rectum. A thick layer of fat in the abdominal wall or great omentum, combined with flatulent distension, has been known to mask entirely an acute abscess caused by perforation of the appendix.

The *functions* of the vermiform appendix are unknown. After it has been removed, no alteration has been observed in the functions of the

alimentary tract, nor in the composition of the blood. It secretes a fluid which is an adjuvant to digestion (Macewen). It is not by any means clear that the appendix is a retrograding organ, although the weight of evidence is in favour of the supposition that it is all that remains of a more capacious caecum. Its lymphoid and other tissues persist until old age. I have seen the follicles but moderately atrophied between eighty-three and eighty-four years of age. According to Dr. Berry the vermiform appendix is specially developed to accommodate the lymphatic tissue accumulated within its walls.

Etiology.—Appendicitis is rare before the fifth year. The age of the youngest patient upon whom I have operated was three and a half years, and of the oldest eighty-three and a half. Both had an acute abscess, and both recovered. But instances of appendicitis in infants from seven weeks of age and onwards have been recorded, and a negro is said to have been born with an inflamed appendix in an umbilical sac (Kelly and Hurdon). Appendicitis is commonest between the age of 10 and 20 years, and next commonest between 20 and 30. Sir Frederick Treves made the following estimate from 452 cases of Hawkins and Fitz:—

Ages.	Proportion of cases per cent.
5 to 10 years	10·8
10 to 20 years	40·7
20 to 30 years	29·0
30 to 40 years	11·5
40 to 60 years	4·6
Over 50 years	3·4
	100·0

This estimate is borne out by Mr. Clogg's, which was based upon 125 cases in Charing Cross Hospital (18). In these the first attack occurred before 10 years of age in 11 cases. Between 10 and 20 in 49 cases. Between 20 and 40 in 45 cases, and after 40 in 10 cases.

Males are more liable to appendicitis than females, but their relative liability is not so disproportionate, as is sometimes thought. The statistics of Mr. Lett (18), which were founded upon 1000 cases operated upon in the London Hospital, give a proportion of about two males to one female (males 684, females 326). Of 200 patients operated upon by myself 118 were males and 82 females.

It is impossible to say whether heredity has any influence, for the simple reason that we possess no ancestral records of appendicitis. But I have operated upon father and son; once upon brother, sister, and first cousin; twice upon brother and sister; and once upon sisters. None of these afforded any evidence of a common developmental peculiarity which might dispose to appendicitis; such, for instance, as the foetal type of appendix, or the pelvic position. Nor was the appendicitis from which these relatives suffered of exactly the same kind. But most medical

men who are able to learn family histories can adduce instances, such as I have given, which lead one to think that the possibility ought not to be lightly dismissed.

Occupation may have some influence. For instance, appendicitis is certainly rather common amongst commercial travellers, and amongst medical men, both being engaged in arduous callings, with much exposure and fatigue, and with irregular meals. Exposure to cold is a frequent exciting cause. It seems reasonable to infer that any rapid cooling of the surface of the body would lead to congestion of the interior, and especially of an organ already inflamed. Thus, attacks have been noted as having followed a railway journey, a funeral, a garden-party, and so forth. Also, as Sir Frederick Treves has pointed out, appendicitis occurs in chronic dyspeptics who either do not or cannot masticate their food. Improper food and improper quantities of food have sometimes been eaten before the beginning of an attack. But, obviously, it is difficult or impossible to say whether these various events originated the appendicitis, or whether they caused an exacerbation of an inflammation already in existence. Dr. Hawkins has adduced evidence to shew that appendicitis is commoner in summer than in winter. It is also said to be commoner in tropical than in temperate climes (Treves).

Injuries are, without doubt, an exciting cause of appendicitis. Attacks have followed blows in the region of the caecum. Strains, too, as in lifting, have preceded attacks. But it is unnecessary to point out that these injuries may have merely increased an appendicular inflammation, which was already in existence. In the same circumstances an injudicious purgative has precipitated an attack. The presence of foreign bodies within the lumen is also an exciting cause. An extraordinary variety have been met with (*vide* p. 599).

Morbid Anatomy and Pathology.—The naked eye is an untrustworthy guide to diseases of the appendix. It has failed to see early ulceration of the mucous membrane, in which bacteria have already begun their invasion. It has confused tuberculosis and cancer; and with the naked eye alone the ray-fungus and *Bilharzia haematobia* would have been overlooked. Clearly every appendix that is removed should be microscopically examined. Statistics based upon naked-eye or mere clinical evidence are quite unreliable. A scientific classification of appendicitis can only be based upon pathology. When that basis is adopted, there is seldom any difficulty in placing the diseased appendix in its proper group. Further, with an adequate knowledge of the morbid anatomy and pathology of appendicitis, the surgeon forms clearer concepts when confronted with clinical cases.

Systematic microscopical examinations shew that ulceration of the mucous lining is present in about 80 per cent of appendicitis. In the early stages, and when the ulcers are shallow, the exterior of the appendix may look perfectly healthy; or its venules may be slightly distended and tortuous; later, the engorgement increases, and the peritoneal covering becomes rough, and also that of the neighbouring ileum

and caecum. At the same time the appendix swells a little, and is harder to the touch and more rigid. But it is remarkable what extensive ulceration may in some cases be going on within the appendix without the slightest alteration of the exterior. This is particularly so when in ulcerative colitis, dysentery, or enteric fever the ulceration spreads to the mucous lining of the appendix. At first the ulceration may be confined to a small part of the mucous lining, but a very little breach admits the invading army of bacteria. Much then depends upon the character of the invaders; some are deadly. All the pyogenetic staphylococci and streptococci, the pneumococcus, many kinds of colon bacillus, the various bacilli associated with putrefaction and gangrene, the typhoid bacillus, the tubercle bacillus, and others may be met with in the diseased appendix. The ulceration begins by the separation of the epithelial lining of the lumen of the appendix, and of the mouths of the tubular glands. So long as it holds out, the epithelium is a perfect barrier against bacterial invasion. But so soon as it is detached, the bacterial inhabitants of the lumen begin to spread along the lymph-canaliculi and lymphatics. The mucosa is crowded with inflammatory cells, and begins to ulcerate. The bacteria also pass into the lymphoid follicles, and into their basilar lymph-spaces and onwards into the submucosa (*vide* Figs. 20, 21). The inflamed tissues of the submucosa become swollen, oedematous, and studded with small haemorrhages. All these help to stretch the muscular and peritoneal coats, and the stretching of these, in whatever way brought about, is one of the chief causes of appendicular pain. But the ulceration of the mucosa is commonly associated with an accumulation of muco-purulent fluid, or of faecal material, within the lumen. Owing to swelling of the mucous membrane, to bends, kinks, torsion, cicatrices, strictures, adhesions, faecal concretions, foreign bodies, or new growths, the lumen becomes obstructed. The discharges from the inflamed and ulcerated mucous membrane cannot escape into the caecum, and begin to accumulate. Or the faecal contents increase by the multiplication of the bacteria of which they are almost entirely composed. For microscopically bacteria constitute much more than the third, which is the normal proportion in faeces (Strasburger). As the lumen becomes distended, its folds disappear and it becomes circular. Then the septic contents pass into the tubular glands, destroying their epithelial lining (*vide* Fig. 19). From these plugged sockets the bacteria make their way into the depths of the mucosa and into the submucosa, and give rise to inflammation or abscesses there. Sometimes, too, the muco-purulent or faecal contents of the lumen can be seen forcing their way into the lymph-spaces and lymphatics opened by ulceration. The lymphoid follicles inflame and swell, and are crowded with inflammatory corpuscles which congregate in their germinal centres, around their margins, and in their basilar sinuses. Septic thrombi are formed in the venules. The septic inflammation started in the mucosa spreads onwards to the submucosa, and by continuity of lymphatics, vessels, and tissues at the muscular hiatuses, to the peritoneal covering of the appendix, to the

meso-appendix, and to the contiguous peritoneum. In all of these the inflammatory changes are alike, but the engorged peritoneal blood-vessels are easily seen and at once arrest attention. The microscope may reveal groups of bacteria amidst the cells which crowd the submucosa, and amidst the cellular exudation which has made the surface of the peritoneum rough and granular. But it is certain that the cells of the peritoneal exudation offer a most determined resistance to the bacterial invasion, and usually bring it to an end. So far the changes have been: first, accumulation of faecal or of muco-purulent fluid in the lumen; second, ulceration and inflammation of the mucosa; and third,

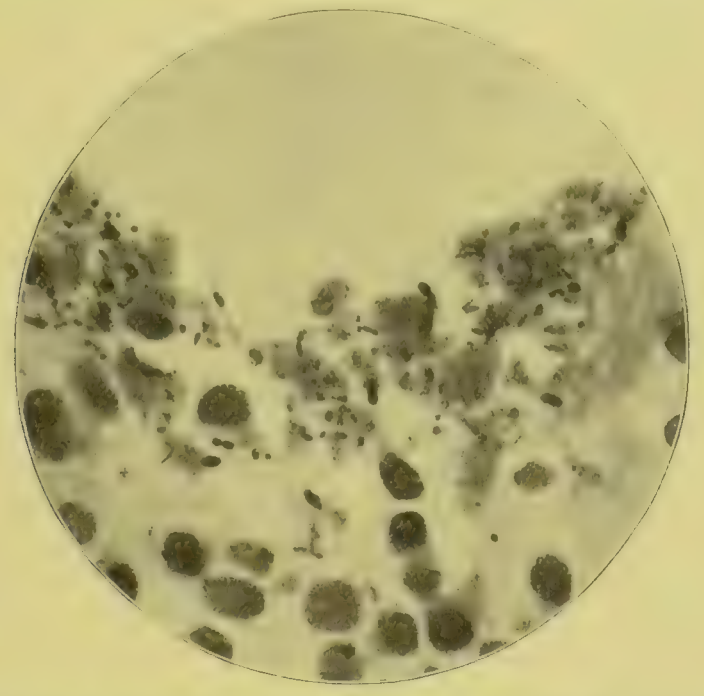


FIG. 21.—From a case of appendicular colic. The mucosa is ulcerated, and its substance crowded with bacteria which have penetrated nearly one-third of its substance. The lumen is above. × 1000.

inflammation of the submucosa and of the peritoneum. During the first stage the clinical signs are slight and often misunderstood. The term appendicular colic is usually applied to them. There is little objection to this term, provided it be not taken to mean a mere painful muscular spasm, and the danger of ulceration and bacterial invasion be not ignored. The course of appendicitis is greatly influenced by the physical and bacteriological properties of the contents of the lumen. When the lumen is unobstructed, we may infer that fluids usually escape into the caecum, and for a time the attack subsides. But very often, when the contents are faecal, the bacteria multiply and collect around a particle of undigested food, and grow into a small, soft, moist lump of faeces, which afterwards becomes drier and harder, and at last calcareous in its centre. Once started the faecal concretion grows bigger by the multiplication of

its bacteria, and its escape into the caecum becomes difficult or impossible, and at last it may distend the appendix and thin its walls, until its dark colour can be seen looming through. When this has taken place, the blood-supply may be mechanically hindered, and sloughing and perforation result. But the concretions which slip out through the hole whilst the appendix is being taken away are usually too small and too soft to have caused the perforation by their pressure; and it seems more reasonable to suppose that the destruction is caused by the bacteria which cover their surface, and which in sections can always be seen invading the ulcerated surface of the mucosa. Some of these bacteria are of the most virulent nature, and when implanted upon the freshly cut abdominal wall may sometimes be seen to cause the most intense inflammation, sloughing, and gangrene. Faecal lumps or concretions are commonly met with in appendices which are obstructed by strictures, kinks, or bends, also those which hang down into the pelvis. Unable to escape into the caecum, they become permanent sources of septic poisoning, the ulceration which is invariably present in the mucous lining allowing absorption by the lymphatics and blood-vessels. Thus, an appendix which has a concretion in it should be removed. In about 2 per cent of cases the faecal contents of the ulcerated appendix are mixed with thread-worms (*Oxyuris vermicularis*). When in abundance, the living worms are easily seen with the naked eye; but when few are present, they may only be brought to light by the microscopical examination. It is doubtful whether these helminths could by themselves cause appendicitis. But in my own cases they have always been mixed with faeces, either fluid or solid, and there has been an obstruction which prevented the mixture from passing into the caecum. *Trichocephalus trichiurus* has been found in the appendix by Mr. James Morrison and others. This comes from the caecum where it dwells. Inasmuch as it pierces the mucous membrane with its whip-like head end, it is likely to be harmful. Round worms (*Ascaris lumbricoides*) and segments of more than one variety of tape-worm have been met with. Many foreign bodies have been found in the appendix. Pins are not very uncommon, glass, hair, bristles, fish-bones, shot, gall-stones, teeth, and, in fact, almost anything that can get into the human intestine. The presence of seeds, and pips, and fruit-stones has been much exaggerated, and must be very rare indeed. As a rule, the foreign body has upon it a layer of faeces, and may be the nucleus of a concretion. There is no clinical method of diagnosing the presence either of concretions or of foreign bodies. As septic bodies they cause both inflammation and ulceration of the mucosa, and sharp or rough ones may excoriate or perforate. Their presence causes continuous pain and tenderness, accompanied with alterations in the pulse and temperature, which are characteristic of septic inflammation in any part of the body. As in other tissues and organs, the bacterial invasion of the walls of the appendix may cause at last an intensely acute inflammation, ending in sloughing or gangrene. Then the appendix is swollen, and its vessels engorged with blood, some of which is usually extravasated

into its tissues: its lumen full of fetid pus, mixed perhaps with faeces, or with faecal concretions: and at the same time its tissues become softer and more easily torn across. A part, or the whole of it, may be black, ashen-grey, bloodless, and dead. When such an intensely inflamed appendix is microscopically examined, its epithelium, tubular glands, and lymphoid follicles have disappeared throughout the whole or part of its extent. Their place is taken by countless inflammatory cells, which also have crowded into the oedematous submucosa, and into the muscular and peritoneal coats. The latter usually has patches of fibrinous lymph upon its surface which may have sealed a perforation, or hemmed in a patch of gangrene. Amidst these inflammatory cells bacteria abound, but sometimes few can be seen, having, presumably, been destroyed by the inflammatory cells. The neighbouring peritoneum, too, is acutely inflamed, and becomes the seat of abscess. It also throws out fibrin oftentimes in great abundance. The meso-appendix, together with its lymphatics, is also inflamed, and its veins may be full of septic thrombi. Such an acute and septic process as this is accompanied by a corresponding degree of local pain and tenderness, together with constitutional disturbance, acceleration of the pulse, and heightened temperature. The same kind of acute infective inflammation can be seen and studied upon the surface of the body. In cellulitis of the finger (paronychia cellulosa), for example, the inflammation, suppuration, death of tissues, and lymphangitis are all in evidence, and cause intense local pain and tenderness, together with acceleration of the pulse and elevation of the temperature. But this series of events in one of the outworks of the body cannot be compared in point of danger with that which takes place in its interior. Far oftener than is thought bacteria reach the outside of the appendix, and infect the peritoneum without perforation, sloughing, or gangrene having taken place. Indeed, it is almost certain that the passage of bacteria is always antecedent to any of these events, with the exception of perforation by sharp foreign bodies such as pins. For instance, a youth was travelling about although he had got appendicitis. The mucosa of his appendix was ulcerated, and an abscess had formed in the submucosa. At this point the muscular coats had disappeared, and there was nothing between the abscess and the general peritoneal cavity but a layer of peritoneum half a millimetre thick, and this was crowded inside and out with bacteria. To find a periappendicular abscess apart from perforation, sloughing, or gangrene, is a common surgical experience.

In addition to bends, kinks, torsions, adhesions, foreign bodies, and concretions, the lumen of the appendix may be narrowed or occluded by strictures. These are caused either by the granulation and cicatrisation of ulceration, or by the formation of cicatricial tissue in the submucous, muscular, and peritoneal coats. Of the first process, which is the same as that seen in any cicatrising ulcer, ulceration of the rectum is, perhaps, an apposite example; of the second, the formation of urethral stricture. Sometimes the circumference of the appendix is not diminished at the seat of the strictures, which are then detected by slitting it open;

or, more accurately, by microscopic sections. But occasionally the appendix is constricted at the stricture, and looks as though a string had been tied round it. When the stricture has followed ulceration, the mucous lining in this situation has disappeared wholly or in part; but when there is a ring of cicatricial tissue in the submucosa, the lumen becomes a narrow canal lined with epithelium, and hard to tell from a tubular gland. Several strictures may form and divide the lumen into compartments, which may or may not possess a mucous lining. But usually there is only one narrowing or occlusion close to the caecum.

The appendix becomes distended beyond the stricture, and the pain

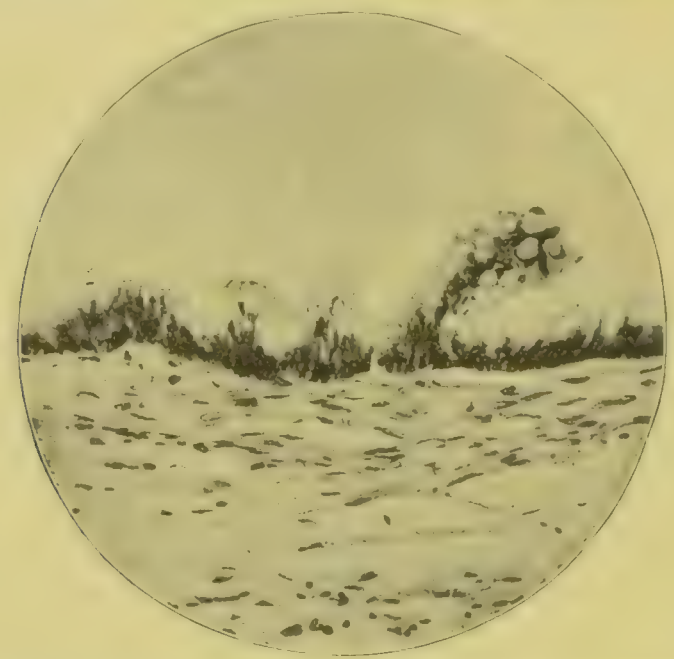


FIG. 22.—The inner part of the wall of a mucocele shewing its lining of goblet-cells. The lumen is above. × 200.

is continuous and severe. Faecal material and concretions are the commonest accumulations, and are accompanied by the usual ulceration and bacterial invasion. Sometimes a starch-like fluid fills the cystic appendix; and although this may have occasional bacteria in it, it does not seem very harmful when it escapes during the operation. Such mucous cysts usually have a complete protective lining of mucous membrane. Or the appendicular walls may be stretched and thinned to translucency by thick transparent mucus secreted by the lining membrane, which consists of a layer of goblet-cells (*vide* Fig. 22). The contents of such a mucocele have been compared to vitreous humour, and are sterile. When the mucocele is loose, and slips about in the abdomen, it is easily and safely taken away without opening the caecum, by merely cutting through the constriction. But oftentimes it is adherent, and bursts while being separated. I have, however, on two such occasions,

seen no ill results whatever ensue from the escape of the sterile fluid; no drainage was required. There is no evidence that mucoceles burst at odd times, and send their mucus amongst the intestines. Or, again, in stricture of the appendix, pus may collect and produce an empyema. Such an appendix is adherent, and may be torn during removal, allowing fetid pus to escape into the abdomen amidst the inflamed and lacerated tissues. When this has taken place, it is safer to insert a rubber drain for a few days. An empyema of the appendix may be tuberculous.

In a small percentage of cases—from 5 to 7 per cent—the lumen of the appendix undergoes complete obliteration. The mucosa is removed by ulceration, and the centre of the appendix filled with a solid core of inflammatory tissue. There is usually nothing upon the exterior of the appendix to shew that obliteration has occurred, although it may feel harder than usual. It might be supposed that, when obliteration has taken place, the appendicitis would subside. But some of those in whom it has been met with had clearly suffered for months or years from great pain, making their lives miserable. A man who had had excessive pain for years, had at the centre of his obliterated appendix a quantity of crystals of what was thought by Dr. F. W. Andrewes to be carbonate of lime. It is certain that obliteration of the appendix does not, as some have supposed, bring about a cure. Unfortunately, it is not yet possible when a patient has appendicitis to divine the pathological condition of the inflamed appendix, and say that it has become obliterated. It may be obliterated, but then, on the other hand, it may contain a concretion, or a foreign body, or faecal matter, or pus, or mucus, which cannot escape, and must of course prevent obliteration. It may be assumed that an inflamed obliterated appendix is not so dangerous as one which is unobliterated.

As a rule, inflammation of the lymphoid follicles and lymphatics is consequent upon destruction by ulceration of the protective covering of epithelium. But in a small proportion of cases—perhaps 3 per cent—the follicles become greatly swollen and inflamed, and their lymphatics dilated and inflamed, although no breach can be detected in the epithelial lining of the lumen or of the tubular glands. Besides the lymphoid elements of the mucosa, the submucous, muscular, and peritoneal coats are inflamed and swollen, but not swollen to such a remarkable degree as the lymphoid tissue. The appendix in this condition is inflamed and adherent, but can be easily separated with the finger. It is twice or thrice its usual diameter, straight or slightly curved, and feels exceedingly solid. The lumen is not enlarged, but is perhaps diminished in size, and may contain epithelium, pus, blood, mucus, and various bacteria, but none of the latter can be seen amidst the inflamed tissues. When no ulceration of the mucosa nor bacterial invasion can be discovered, the cause of the lymphadenitis becomes obscure. There is no evidence whatever that it is tuberculous. All the lymphatic tissues of the body are subject to inflammatory swellings, the causes of which are often unknown. Much work remains to be done at this branch of pathology.

Lymphadenitis and lymphangitis of the appendix are met with in children and young adults. I have seen them accompanied by general enlargement of the lymphatic glands. The attacks are characterised by an unusual amount of pain, but do not seem to be particularly dangerous because, usually, several have been endured before the curative operation.

Tuberculosis of the appendix is met with in 2 per cent of operation cases, and is therefore rather rare. An accurate diagnosis cannot be made without a microscopic examination. Nodules which to the naked eye looked like miliary tubercle, proved to be merely inflammatory, and others which looked tuberculous were cancerous. In the appendix, as elsewhere, the tubercle bacillus causes inflammation followed by the

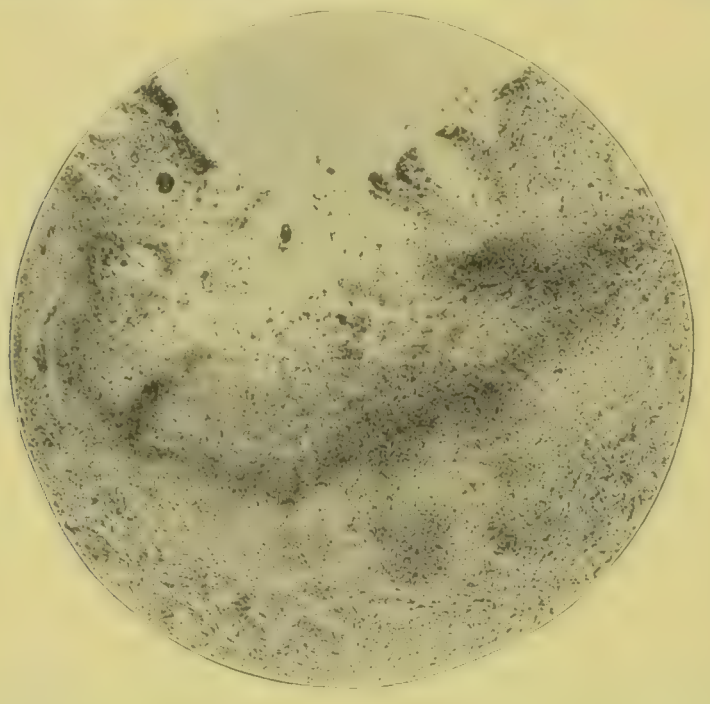


Fig. 23.—Tuberculous appendicitis. A tuberculous abscess or ulcer opening into the lumen. ×50. The lumen is above.

characteristic caseation, and subsequent softening of the caseous material into pus, and the formation of ulcers upon the mucous membrane (*vide* Fig. 23). In those who are suffering from tuberculous ulceration of the upper air-passages or of the lungs, the bacilli may be swallowed, and so reach the lumen of the appendix. In such circumstances there is usually tuberculous ulceration throughout the alimentary tract, and the question of operative treatment can scarcely arise. The ulceration of the mucous lining of the appendix may only be discovered at the necropsy, having been unsuspected during life. But oftenest the tubercle bacillus may be carried to the appendix in the blood-stream, the mode of ingress being unknown. In these cases tuberculosis is seldom confined to the appendix, but is spread about the peritoneum along the peripheral distribution of the superior mesenteric artery. In these circumstances it is difficult to say

whether the appendix and peritoneum were attacked simultaneously, or whether tuberculosis began in the appendix and spread to the peritoneum, or vice versa. From the appendix tuberculosis usually spreads to the neighbouring glands and to the caecum, and the ascending colon may also be tuberculous and undergo thickening and cicatrisation. The pus which is the result of the tuberculous process may accumulate in the interior of the appendix and form an empyema; or around the appendix and beneath the caecum. I have observed these tuberculous abscesses pass down beneath the crural arch and point at the outer part of Scarpa's triangle; and also that appendicular abscesses which track down the thigh are usually followed by a faecal leak. To cure this formidable condition it may be necessary to resect the end of the ileum, the caecum, and right colon, together with the appendix and tuberculous glands. The tuberculous origin of an attack of appendicitis is to be suspected if the patient be suffering from tuberculosis elsewhere; as, for example, in the lungs. The estimation of the opsonic index of the blood is now under trial as an aid in the diagnosis of tuberculosis. Although one may suspect that tubercle coexists in the peritoneum or elsewhere, the urgency of the appendicular symptoms may necessitate appendicectomy. The ultimate result is better than might be expected, especially when the tuberculous peritonitis is of the kind in which there is fluid in the abdominal cavity. But the outlook is most unfavourable when it has caused adhesions and quantities of caseating inflammatory thickening. In this class an injudicious operation usually ends in a faecal leak, and if silk be used it becomes tuberculous, and has to be extracted, so that catgut should be used.

The systematic microscopical examination of the excised appendix brings primary carcinoma to light with rather ominous frequency, the proportion being perhaps not far short of 1 in 250 or 1 in 300. The diagnosis cannot be made with the naked eye; for with that alone the growth has usually been mistaken for tuberculosis, or for an ordinary inflammatory nodule. Sometimes it is impossible to tell whether the carcinoma began in the appendix and spread to the caecum and ileo-caecal valve, or the reverse. The commonest form of carcinoma has in its stroma cells of approximately spheroidal shape, and is usually referred to as spheroidal-celled carcinoma. Columnar-celled carcinoma is also found, and may be undergoing colloid change (21). When inflammatory obliteration of the appendix is taking place, the remains of the tubular glands sometimes simulate columnar-celled carcinoma. But this ought not to deceive, because the groups of cells radiate from the axis of the appendix, just as the tubular glands radiate, and the appearances are usually seen near the distal end. Up to the present, carcinoma of the appendix can hardly be said to have been diagnosed during life. It causes symptoms which cannot be distinguished from an ordinary attack of appendicitis. On one occasion cancer was suspected on account of the family history of cancer; and on another on account of the marked anaemia, wasting, and loss of energy. Cancer of the appendix may be

met with at any age from youth upward. Left alone it runs the ordinary course, infecting the lymphatic glands and spreading to the caecum, small intestines, and peritoneum, and causing abscesses and fistulous openings. The prognosis is favourable after early and complete removal. Other types of malignant growth, such as sarcoma and endothelioma, have been reported. Secondary deposits of carcinoma also occur in the appendix. Sometimes the primary growth is in the ovary. Primary and secondary sarcomas have also been found in the vermiform appendix. I have seen sarcoma of the appendix secondary to sarcoma of the chest-wall. Fibromas and polypoid growths have also been recorded.

The vermiform appendix, caecum, right colon, ileum, together with the neighbouring peritoneum and abdominal wall, are occasionally the seat of actinomycosis. The inflammation which the ray-fungus sets up joins those structures together into a hard mass, which softens and ulcerates. Usually the nature of the disease is not suspected until an abscess has been opened, or has burst and brought to light the characteristic yellow specks composed chiefly of mycelium, but sometimes of mycelium and clubs. Or, after the abscess has been opened, suspicion may be aroused by the persistence of the induration and suppuration, and by the formation of fresh sinuses or faecal leaks. Then the streptothrix or mycelium may be sought for and found. The recognition of the exact nature of the disease is of extreme importance. It may be cured or greatly alleviated by large and continuous doses of iodide of potassium, together with the erasion and cleansing of the sinuses to prepare them for abundance of iodoform in fine powder and in emulsion. An attempt to resect may be undertaken as a last resort, the presence of a faecal leak compelling interference and justifying extreme measures. In the inflamed appendix of a man from South Africa I found the eggs of *Bilharzia haematobia* (5).

The Peritonitis.—A time comes when the appendicular inflammation spreads to the peritoneum. The dangers of this complication are appreciated when we recall the anatomy of that great serous membrane; its vast superficial area; its close relations with the stomach and intestines, of which it may be considered an integral part; its close relations with the other abdominal viscera; its nerve-supply from both somatic and splanchnic systems; its abundant vascularity; and, above all, its countless stomata opening into numerous lymphatics. In addition, the peritoneum is a sac without exits for the escape of septic fluids. Its surfaces, too, are continuously gliding upon one another, owing to the respiratory movements of the diaphragm and abdominal walls and the peristaltic movements of the intestines. Once started, an infective inflammation of the peritoneum has an enormous area over which it may travel, and few natural anatomical barriers to arrest its progress. Its onward course, too, is aided by the continuous involuntary movements, and the bacteria which cause it, together with their toxins, unable to escape, enter the veins and lymphatics. Fortunately, the peritoneum is endowed with extraordinary powers of adhesive inflammation and of phagocytosis, so

hat abscesses become hemmed in and localised, and bacteria killed and aten up.

In some degree the type of the peritonitis conforms to that of the ppendicitis from which it originates. When that is mild, as, for instance, vith superficial ulceration of the mucosa and bacterial invasion, the eritoneum of the appendix itself, of the meso-appendix, and of the eighbouring intestines is slightly engorged with blood, and roughened, ogether with a curious shagginess of the end of the ileum and caecum. The peritonitis, too, is slight in cases of mucocele or of fibrous oblitera- ion. But when the mucosa is deeply ulcerated, the lumen distended vith virulent contents, and the inflamed appendicular walls teeming with acteria, the peritoneum is intensely engorged with blood, swollen, overed with patches of lymph, infiltrated with cells, and its lymphatics nd lymphatic glands inflamed. At last some fluid collects and soon ecomes purulent. Such changes as these may, as we have seen, be set p without any actual perforation or gangrene of the appendicular walls. n perforation or gangrene of the appendix the peritonitis is of the cutest and most dangerous type. But even then the resisting powers of he peritoneum often prevail. Whilst operating it is not unusual to have o undo many adhesions, and at length find in their midst a perforated ppendix surrounded by fetid pus, which may have a faecal concretion n it. When the appendix is gangrenous such barriers of adhesions are ess common, for it seems as though bacteria which can kill the appendix an overcome the resistance of the peritoneum and cause a diffuse septic eritonitis.

The position of the appendix at the moment when it perforates or ecomes gangrenous has considerable influence upon the formation of dhesions, and upon the extension of the inflammation. Thus, the ileo- aecal junction is a favourable spot for the formation of limiting adhe- ions, also underneath the caecum, or external to the caecum and colon, r deep down in the pelvis. When the herniated appendix gives rise to uppuration within the ileo-caecal or retro-colic fossa a definitely localised etro-colic abscess is the result. But in severe cases of diffuse septic eritonitis the gangrenous appendix is often encountered amongst the oils of the ileum and floating about in thin pus. The pus in diffuse eptic peritonitis collects amongst the coils of the small intestines, in the elvis (the great receptacle for abdominal effusions), in the iliac fossae, nd in the flanks. The transverse mesocolon, transverse colon, and reat omentum together form a partition which keeps it out of the upper art of the abdomen. But sometimes it passes round to the right, and ollects in the kidney-pouch described by Mr. Rutherford Morison. These oints must be considered when the operator endeavours to remove the us and provide efficient drainage. Occasionally the peritonitis leads o the formation of a considerable quantity of thin, clear fluid, which ins out when the incision is made. Mr. W. G. Spencer has recorded good example of recurrent appendicitis complicated by general serous eritonitis and effusion. The presence of serous effusion suggests the

presence of tuberculous peritonitis, but I have more than once seen it when no evidence whatever of tuberculosis could be obtained. Further, small, grey nodules may be seen about the appendix and peritoneum, but are not necessarily of tuberculous origin, for some are merely inflammatory, and the subsequent history of the cases is quite favourable.

The septic inflammation may cause great thickening of the parietal peritoneum, and this may be over the site of the localised appendicular abscess. This thickened and engorged serous membrane is crowded with leucocytes and fibrinous exudation. The thickening may be as much as half an inch. The intestinal peritoneum is also thickened, but not in the same degree, and is closely adherent to the parietal. In these circumstances an attempt to reach the abscess by cutting through the thickened peritoneum may wound the bowel. As the thickened peritoneum feels very hard and solid, it is best to search for a soft place to incise, and, failing that, to begin at the edge of the hardness and work towards the iliac fossa. Should the peritoneal cavity be opened, a gauze dam should be arranged before the finger is pushed into the abscess.

A localised collection of pus in the iliac fossa is the commonest form of appendicular abscess, and the easiest to diagnose and to deal with. Localised collections also form in the recto-vesical or in Douglas's pouch (called in future a pelvic abscess); or to the right of the ascending colon, and bulging into the right flank (called in future extra-colic); or behind the right colon (called in future retro-colic). Each of these has distinctive peculiarities of its own. A pelvic appendicular abscess is, as a rule, associated with the pelvic position of the vermiform appendix, which occurs in 17 per cent of operation cases. But a pelvic abscess may form although the appendix is in the iliac fossa. I have opened an iliac and a pelvic abscess simultaneously. The intestines and great omentum are usually matted together and form a dome over the pelvic abscess, which, as it increases, lifts them up, and may ultimately burst into the general peritoneal cavity, or, more fortunately, per rectum. In a neglected case a distended bladder may be closely simulated. Below, the pelvic abscess can be felt bulging into the rectum, and in women into the posterior fornix of the vagina. In cases of pelvic appendicular abscess the pelvic peritoneum is acutely inflamed, so that digital pressure per rectum is intensely painful. The pain, too, may be sharp when the bladder (or the rectum) is emptied. Occasionally the pelvic abscess does not form until after an operation, at which perhaps a septic appendix has been removed and an iliac abscess emptied and drained. If, therefore, in such circumstances the pulse and temperature should not fall, the pelvis should always be examined. As a rule, the pelvic abscess is emptied and drained through an abdominal incision, usually made in the right semilunar line, for this affords an opportunity of removing the appendix. I have also opened pelvic abscesses per vaginam and per rectum, and the results have been very favourable. From the omission of rectal examinations many pelvic abscesses have been over-

ooked, and oftentimes with disastrous results. In extra-colic appendi-ular abscesses the septic appendix usually lies to the outer side of the aecum, so that the pus spreads upwards and towards the right into the lank. More rarely, the appendix itself springs from the right-hand side of the caecum, and stretches upwards along the right-hand side of the scending colon. When such an one gives rise to suppuration it usually appens that, as the abscess grows, it bulges the right flank, which is lull on percussion and becomes slightly convex instead of concave like he left. Such an extra-colic abscess is emptied and drained without pening the general peritoneal cavity by an oblique incision an inch or o above the middle third of the iliac crest. A long appendix running pwards behind the right colon in the ileo-caecal or retro-colic fossa may ive rise to a retro-colic abscess in two ways. The appendix itself may ecome distended with pus. I have mistaken such a retro-colic empyema of the appendix for a small pyonephrosis, and the lower part of the ppendix for the ureter. Or, much more commonly, the pus accumu-ates outside the septic appendix and within the retro-colic or the ileo-aecal fossa. At this stage a hard, tender, inflammatory mass is felt eneath the right linea semilunaris. There is usually a resonant area ll round this tumour. I believe that ultimately the pus perforates the iliac fascia, and follows the iliacus muscle into the thigh. This retro-olic abscess is opened by entering the abdomen by the usual oblique ncision, and then incising the peritoneum outside the colon as if to get t the right kidney. It is drained through a separate opening in the ight flank. It is probable that as the diagnosis becomes more certain nd accurate, surgeons will adopt a lumbar incision for retro-colic abscess, nd thus avoid opening the general peritoneal cavity. In neglected ases the pus from appendicular abscesses oftenest bursts through the bdominal wall, or less seldom into the caecum. But it has also escaped nto the bladder, ureter, and into the great arteries and veins of the elvis. It has tracked along the psoas muscle (Barnard). Statements hat the stools or urine contained pus are to be received with caution, nd not accepted without a microscopical examination. Sometimes the us in localised abscesses seems to form a favourable culture medium for particularly noxious species of bacillus. Four times I have seen acute angrene of the muscular planes of the abdominal walls after the evacua-ion of localised appendicular abscesses, and in three instances it proved atal. We have here an additional reason for the immediate evacuation f the pus. The pus of appendicular abscesses contains gas-forming acilli, so that they often contain a good deal of gas, and are ympanitic. Also these intestinal bacilli may convert the pus into a luid which in smell and appearance closely resembles fluid faeces. The presence of faeces in abscesses should not, therefore, be assumed vithout a microscopical examination, nor, as a rule, the presence of a aecal leak.

As might be inferred from the source from whence it arises, the nfection of the peritoneum is nearly always of the mixed variety. In

the less severe kinds of non-suppurative, localised peritonitis, cocci, diplococci, encapsuled cocci, streptococci, and bacilli of various shapes and sizes may be seen amidst the little heaps of inflammatory cells and fibrin upon the surface of the peritoneum. In the more acute kinds, in which suppuration is impending or has begun, the exudation swarms with bacteria, but with a great preponderance of one morphological variety. My observations have led me to think that the bacteria of the colon bacillus group and its congeners do not easily penetrate the peritoneum, but flourish best amidst the surface exudation and pus, and produce their foul gases and toxins. This form of peritonitis is much more amenable to surgical treatment than might be supposed, but, unfortunately, virulent streptococci may be present and penetrate the lymph-spaces and lymphatics of the peritoneum, causing a swiftly spreading and fatal peritonitis. This streptococcic peritonitis is to be suspected when the intensely inflamed peritoneum is bathed in thin, odourless pus, and has few or slight adhesions. We still need better methods of diagnosing this dangerous form of infection in its earliest stages, and before it has got beyond the influence of antistreptococcic serum. Besides streptococci, the colon bacillus may be associated with *Pneumococcus*, *Staphylococcus pyogenes albus* and *aureus*, *Bacillus pyocyaneus*, *Bacillus aerogenes capsulatus*, and many others, aerobic and anaerobic. It is unusual to obtain pure cultures of these bacteria from cases of appendicitis. There is, as I have just said, undoubtedly imprisoned in some small localised appendicular abscesses a bacillus which, when set free by the operation, causes acute spreading gangrene of the abdominal walls, accompanied with the most horrible fetor.

Clinical Signs.—*Commencing and Chronic Appendicitis.*—The clinical signs of appendicitis depend upon the kind of inflammatory action going on in the appendix itself, in the end of the ileum, the caecum, ileo-caecal valve, and adjoining structures, especially in the peritoneum; and upon the general constitutional disturbance and alteration in function caused by inflammation. Generally speaking, the constitutional symptoms are the same as those which occur in septic inflammations of other organs or parts of the body. It is convenient and fairly correct to group cases as chronic, subacute, and acute: the chronic may be taken to include those which are commencing and likely to become subacute or acute. For at one time the inflammation is moderate in degree, and continues for weeks, or months, or years; at another, its progress is measured by days or by hours. Such is the nature of this treacherous disease that chronic changes become acute, and acute become chronic; or the inflammation subsides and leaves no trace behind, except perhaps a little pigmentation of the mucous lining of the appendix: or it may fulminate and end with appalling suddenness. At the beginning of acute attacks physicians and surgeons of great experience have erred in their forecasts, so that the greatest watchfulness must be employed lest the signs of danger be overlooked and the golden opportunity lost. A sudden acute attack without premonitory symptoms is greatly to be dreaded, especially in the young;

at the other extreme of life inflammatory changes are slower, and their clinical effects less violent.

The commonest sequence of pathological events in the appendix is, as we have already seen, (*a*) septic accumulation within the lumen; (*b*) inflammation and ulceration of the mucosa; (*c*) bacterial invasion of the mucous, submucous, peritoneal, and muscular coats; (*d*) the passage of toxins, and ultimately of bacteria, into the blood; (*e*) the extension of the septic inflammation to the peritoneum, lymphatic vessels, lymphatic glands, venous system, and adjoining structures. When the septic accumulation and the ulceration of the mucosa are beginning, the symptoms they produce may be overlooked or attributed to other causes; especially, as in the earliest stages, the pain is nearly always felt towards the centre of the abdomen. Days, weeks, or months may elapse before the pain actually "settles" in the right iliac fossa; but when it has settled there the patient points to the spot with one of the most typical gestures of disease. The fingers of the right hand are directed to M'Burney's point, and, at the same time, the right thigh is slightly bent at the hip joint. The early pain of appendicitis is often mistaken for ordinary colic, and, therefore, not thought indicative of danger. If the colic be felt about the right iliac fossa, it is often called "appendicular colic." There is no reason why this term should not be used, provided the pathological changes going on in the appendix be realised (*vide* Fig. 21, p. 599). But when the appendix occupies the pelvic position, the pain may never settle in the right iliac fossa, but continue in the region of the umbilicus. A rectal examination is a routine step in all doubtful cases of abdominal disease, and when the appendix is within the pelvis there is tenderness on pressure in the recto-vesical or recto-uterine pouch, or an inflammatory swelling may be felt there. Other clinical signs may suggest the pelvic position of the appendix, such as pain when the bladder is emptied or distended, pain on defecation, or, in more acute cases, there may be retention of urine. Children may retain their urine, being afraid of the pain during micturition. Sometimes menstruation is accompanied by unusual pain and discomfort, and is likewise excessive. Whilst making the rectal examination, it is important to note the presence or absence of tenderness upon the right side of the pelvis. It will be found present in nearly all cases of appendicitis, although there may be no tenderness whatever at M'Burney's point. This tenderness at the right side of the rectum is a diagnostic sign of great value, but one which is usually overlooked. Not infrequently a vaginal examination is necessary, especially when an inflammatory affection of the uterus and of its appendages or a haemato-salpinx has to be eliminated. In the early stages of chronic appendicitis, digestive troubles are not unusual. The patient may be unable to eat certain articles of diet, especially fruit and vegetables. Indigestion, gastric pain, and occasionally vomiting may be present, so that the case may be diagnosed as one of gastric catarrh or of gastritis. In chronic appendicitis unpleasant odours and tastes are at times experienced,

caused, it may be assumed, by absorption from the ulcerated appendix. When ulceration and bacterial invasion are in progress, slight but obvious changes take place in the pulse-rate and temperature. To obtain a correct record, the patient must be continuously in bed, and the record made every six hours. A rise in the pulse-rate and temperature is often accompanied by a simultaneous accession of pain and tenderness in the right iliac fossa. Shivering attacks, or even a rigor, may supervene. This is a danger-signal of great import, as it probably indicates the presence of virulent pyogenetic organisms. The patient may be thought to be suffering from malaria. Sometimes when the pain has entirely departed, and the pulse-rate and temperature have become normal, the persistence of the tenderness has led me to remove the appendix. More than once in such circumstances the inflammatory changes have been very marked and far beyond what might have been anticipated. To elicit tenderness the patient should be recumbent, with the thighs flexed upon the abdomen and the abdominal wall relaxed. The pressure should be made gradually with the tips of the finger, and end with a little sudden push. The patient often thinks that the pain which is felt was caused with the finger-nail. It is an invariable rule to begin upon the left side, and gradually draw near the region of the appendix. Should no tenderness be elicited at the right iliac fossa it is to be remembered that the appendix may occupy an abnormal position. Tenderness may be masked by flatulent distension of the intestines or by thick layers of fat. Perhaps the tenderness which accompanies the various kinds of colitis is oftenest confused with that of appendicitis. Whilst applying digital pressure, the protective rigidity and spasm of the abdominal muscles is to be noted. A rigid band of muscular fibres is sometimes mistaken for the appendix, as is also the outer edge of the right rectus abdominis. In difficult cases a day or two in bed relaxes the abdomen, and renders the examination easy. The diseased appendix is seldom felt. Oftentimes a thing which is tender, hard, round, and which slips away from the finger can be felt in the iliac fossa, although the appendix is adherent in the pelvis or in some other situation where it could not have been felt.

As a rule, in commencing and chronic appendicitis, the functions of the alimentary tract are in some way disturbed, and yet I have seen the bowels act quite naturally in spite of the presence of an acute appendicular abscess. The action of the bowels may be irregular, constipated, or even loose. The motions may be highly offensive, and contain undigested food, mucus, blood-stained mucus, or blood; for in appendicitis of any degree of severity the mucous membrane of the neighbouring ileum, caecum, and ileo-caecal valve may be inflamed. Sometimes the abdomen becomes distended, so that the clothing has to be undone. This distension may betoken the onset of an acute attack. Also, flatulent rumblings and gurglings may be heard in the intestines. Pressure upon the caecal region may cause a sudden gurgle, due, probably, to the relaxation and dilatation of the caecum which often accompanies chronic appendicitis.

Errors in diagnosis would seldom occur in doubtful cases if the patient were placed under proper observation. It would be easy to give a long list of those which have been cleared up by a week in bed, a dose or two of castor oil, and a record of the pulse and temperature every six hours. If after a period of observation all the symptoms subside, then the patient should be tested by allowing movement and exercise, and by improving the diet. It is reassuring if the pulse and temperature remain steady, and if there be no return of the pain, tenderness, or rigidity. The hysterical can mimic appendicitis with strange fidelity. A period of probation sets doubt at rest and prevents error. It is to be remembered that appendicitis may coincide with hysteria. An inflamed appendix with ulceration and bacterial invasion of the mucosa was removed from a hysterical young lady, who afterwards alarmed those in attendance by mimicking an attack of peritonitis.

Appendicitis is to be diagnosed with extreme caution in those who are known to suffer from gout, for they may have all the ordinary signs of chronic or the ordinary signs of subacute appendicitis, and be quickly relieved by calomel and colchicum. I have known a gouty person to suffer from pain like that of appendicitis months after the excision of the appendix.

Subacute and Acute Appendicitis. — When the appendix is more intensely and dangerously inflamed the pain at the centre of the abdomen may be very severe, and cause the patient to cry out or even faint. At the same time the contents of the stomach are usually vomited up, and afterwards bile from the duodenum. This may be called a "bilious attack," and, as "bilious attacks" are not considered dangerous, calamities have resulted. In the presence of such clinical signs an abdominal examination might shew that there was some general distension, with tenderness and muscular rigidity in the region of the appendix. Or even an inflammatory swelling might be seen or felt there. Furthermore, per rectum, a tender inflammatory swelling might be discovered. The pelvic position of the appendix is a trap for the unwary, and the omission of rectal examinations has cost many lives. At the same time the pulse and temperature ought to be taken, as the pulse is accelerated and the bodily temperature raised. A rigor during the course of any inflammation usually implies a severe and dangerous kind of infection. Appendicitis is no exception to this rule, for in it a rigor is an ominous sign. It is not difficult to imagine the danger when the acutely inflamed organ is inside the largest serous sac in the body, and when it is teeming with streptococci or other virulent bacteria. During acute and severe attacks the functions of the alimentary tract are usually much disturbed. There is great distaste for food. Constipation and flatulent distension are common features. The stools should be examined for mucus and blood. A moderate degree of leucocytosis may be found, 13,000 to 15,000 or 20,000 white cells per c.mm. An advancing leucocytosis is a danger-signal of great import. The abdominal tenderness is greatest in the neighbourhood of the appendix, but all the parietal

peritoneum of the right and lower abdomen, including the pelvis, is oftentimes exquisitely tender. It is wise to mark out, when possible, in aniline pencil the limits of the tender area, as its increase and diminution help to shew which way the disease is progressing. Micturition and defecation may be painful, because the inflamed pelvic peritoneum is stretched when the bladder or rectum is emptied. The pain may be so acute that a child may utter a shrill scream. Similar pain may ensue when either the rectum or the bladder becomes distended. To protect the tender peritoneum the abdominal muscles become rigid and the respiratory movements lessened or abolished over a part or the whole of the abdomen. There is usually flexion of one or both thighs. Vomiting usually occurs once at the beginning of the attack, but now and then it recurs, and may become an alarming and troublesome feature in the case. Hiccup, too, is by no means rare. In most acute attacks of appendicitis the inflamed intestines cease to propel their gaseous and faecal contents, and as the intestinal bacteria continue to make abundance of gases, the abdomen soon becomes distended. An inflammatory swelling may be felt in the right iliac fossa, in the pelvis, the right flank, or behind the right colon, or elsewhere. The inflammatory swelling may be the appendix itself, swollen or distended, and surrounded by thickened peritoneum, intestines, or omentum; or it may be an abscess with its inflammatory surroundings. The size of the inflammatory tumour may be augmented by faecal accumulation in the caecum. Little is to be inferred from inability to find an inflammatory swelling. None may exist, or it may be situated where it cannot be felt, as, for instance, near the right sacro-iliac synchondrosis, or it may be concealed by muscular rigidity, flatulent distension, or adiposity. In thin subjects the inflammatory swelling may project enough to be seen, or it may have lessened the inguinal groove, or the hollow of the flank. An acute inflammatory swelling, which can be seen, nearly always contains pus.

The course of an acute attack of appendicitis can be inferred from the local and general signs. To prevent delay in operative measures acute symptoms should be noted almost hour by hour—certainly four times in the twenty-four hours. Continuous and severe pain; increasing abdominal tenderness and rigidity; augmentation of the size of the inflammatory swelling (should one be found); a persistently high temperature, and, more especially, one which tends to rise rather than fall; a rapid pulse; a pulse-rate of one hundred is a reason for anxiety, and a further acceleration is oftentimes a reason for surgical interference. The human heart is more sensitive than the clinical thermometer. A fall in the pulse-rate is often the first intimation that the inflammation is subsiding; on the other hand, a rising pulse-rate is usually a clear sign that it is spreading. A rapid pulse and a low temperature are of grave import. A leucocytosis which is high and advancing also indicates that the inflammation and sepsis are advancing and not retreating. Other symptoms, which I have elsewhere called "determinant," may supervene. Intestinal obstruction, with its accompanying abdominal obstruction, is

by no means rare; vomiting is occasionally severe and uncontrollable; the pain may be of the most violent and agonising nature. A sudden and acute attack of appendicitis may seem to subside, and be followed by an interval of calm or repose, during which the pain and tenderness decrease and the pulse and temperature fall. For instance, a very stout lady was seized with a sudden and violent attack of appendicitis. The pain, vomiting, and constipation were of the severest type; the pulse 120; the temperature 103° F. The whole of the lower abdomen was tender, and also the pelvic peritoneum. Inasmuch as the heart was fatty, and its action feeble and irregular, an operation during the acute attack was full of peril. Next day, the pulse and temperature fell, and the pain and tenderness almost entirely disappeared. Two days later all the symptoms recurred, and a recent abscess was found beneath the caecum, and around a recently perforated appendix.

The course of appendicitis is most difficult to judge in those who are stout. The tenderness is masked by the fat, and owing to the apparent absence of tenderness, their appendicitis is frequently overlooked, especially when a rectal examination is omitted or impracticable. The apparent improvement not infrequently follows a purge; that which is caused by morphine could hardly lead to error. The pulse is the most reliable of the general signs, but a naturally slow pulse does not easily accelerate, and may never exceed ninety beats a minute, although the appendix be gangrenous and surrounded with pus. A heart that is diseased or hypertrophied does not easily beat at a quicker rate, in whatever part of the body the septic inflammation may be.

In ulcerative appendicitis there is always danger lest the bacteria in the coats of the inflamed appendix should enter the veins, and passing inwards to the liver give rise to hepatic or subphrenic abscesses. The bacteria, too, may be carried onwards by an embolus. When the hepatic abscesses are multiple, the outlook is most unfavourable. The portal pyaemia is usually caused by virulent streptococci. But a single abscess may develop. A favourite site is in front, beneath the upper third of the right rectus abdominis, and near the falciform ligament. In this situation I have operated with complete success in three cases in which the fetid pus contained the less virulent forms of intestinal bacteria. The diagnosis of hepatic or of subphrenic abscess is not difficult, when an acute appendicitis is followed by pain, tenderness, and swelling in the region of the liver. But when the antecedent attack of appendicitis has been overlooked or, as in the "masked" cases, described by Sir Frederick Treves, the inflamed appendix, although full of pus, has caused no symptoms whatever, the diagnosis becomes exceedingly difficult. In a case of my own, an unsuspected periappendicular abscess was found after a man had died of subphrenic abscess and portal pyaemia. Maydl has given similar instances. Sometimes infective inflammation starting in the appendicular and meso-appendicular veins causes pylephlebitis with partial or complete thrombosis of the superior mesenteric vein, and the clot may extend into the portal vein, or into the splenic. The following is an instance of

partial thrombosis. A man with acute appendicitis had thrombosis of the branches of the veins of the last nine inches of the ileum. The intestine and neighbouring mesentery were deep purple in colour, almost black, greatly swollen and oedematous, and looked on the point of gangrene; but, when the intestine was squeezed, it became paler, and some circulation was present in its coats. In consequence, resection was not performed, and the patient recovered. In Elliott's case forty-eight inches of intestine were removed, and secondary suture successfully achieved. In another case of my own, the clotting involved the whole of the superior mesenteric vein, and probably the main portal trunk, and the patient died. In these cases of portal thrombosis the appendicular origin is often overlooked. A systematic microscopical examination of the appendix should be made in all cases of portal thrombosis. The clinical signs of pylephlebitis, or of portal thrombosis, are most ambiguous. In one instance an intussusception had been diagnosed; in another, an acute mechanical obstruction; and, in yet another, a volvulus was suspected.

The bacteria may not be arrested in the liver, but pass onwards to the lungs and pleurae, to the serous or synovial membranes, to the parotid gland, the testicles, or to the brain. The septic pneumonia or septic pleurisy may end in abscess. Treatment by resection of ribs and free drainage affords a fair prospect of success.

During the course of appendicitis, clotting occasionally takes place in the iliac veins—either in the right or in the left. Although this complication is very troublesome, yet it seems to be of comparatively small danger to life, and after months or years have elapsed the limb recovers.

The lymphatic glands which become swollen and inflamed during the course of appendicitis hardly ever suppurate. Their presence within the abdomen and pelvis is usually to be inferred; but not infrequently they are to be felt along the right crural arch, and now and then along the left. The pain and tenderness caused by inflammation of the lymphatic glands behind the right meso-colon resemble the pain and tenderness of cholecystitis or of duodenal ulcer.

When appendicitis causes acute abdominal abscess, the signs are those of acute abscess elsewhere, with special abdominal symptoms superadded. The local signs of this dreaded complication, which, even after operation, is accompanied by a mortality of at least 1 in 10, depend upon the situation of the abscess. The pain is usually severe and continuous; and the tenderness is greatest when the pus is near the parietal peritoneum lining the front of the abdomen, the flanks, or pelvis. The chief general signs are a continuously high temperature, an accelerated pulse-rate which tends to quicken, and an advancing leucocytosis. A man with acute localised appendicular abscess had a temperature of 101·6° F., pulse 80 to 92, and a leucocytosis which had risen in less than forty-eight hours from 21,800 to 35,600. The position of the abscess is indicated by a painful, tender, hard swelling, gradually and continuously increasing in size, and situated (*a*) in the iliac fossa, (*b*) in the pelvis, (*c*) in the right flank, (*d*) behind the right colon, (*e*) or elsewhere, as, for instance, in the

umbilical region. An abscess with its inflammatory surroundings is usually dull on percussion, but too much reliance should not be placed upon this, because the pus contains bacteria which have remarkable powers of gas-production. Abscesses at the ileo-caecal junction, beneath the caecum, or in the ileo-caecal or retro-colic fossae tend to become localised, and may soon cease to increase. Fluctuation cannot be felt unless the abscess has been allowed to attain a considerable size. The attempt to elicit fluctuation is not free from risk of inducing rupture of the abscess into the general peritoneal cavity. In neglected cases the abdominal wall may become red and oedematous, and ultimately give way. An abscess which has been correctly diagnosed can oftentimes be opened without disturbing the general peritoneal cavity. The flank abscess is the most favourable, and then one in the iliac fossa, near Poupart's ligament. An abscess farther in beneath the caecum or right colon, or beneath the ileo-caecal junction, cannot, as a rule, be opened without exposing the general peritoneal cavity, but the pus may, nevertheless, be evacuated with very little soiling and the cavity effectually drained. Acute abscess on the pelvic brim or in the pelvis is less favourable, owing chiefly to difficulties in providing drainage. A retro-colic appendicular abscess simulates rather closely an inflammatory renal swelling. An empyema of an appendix in the retro-colic fossa has been thought to be tuberculosis of the right kidney. In a second instance, the diagnosis of retro-colic abscess was easy, because a characteristic acute attack of appendicitis was followed by the formation of a hard, tender, ovoid swelling beneath the middle third of the right linea semilunaris; a third case of retro-colic abscess was thought to be a malignant tumour of the colon.

In stout subjects, or in those with thick and muscular abdominal walls, small appendicular abscesses cannot be felt, especially when situated near the right sacro-iliac synchondrosis. Ordinarily, the abscess is hemmed in by inflammatory exudation or adhesions, and by gluing together of intestines, great omentum, and abdominal organs. The great omentum usually wraps itself round the inflamed appendix, and helps to shut in abscesses. After the localisation has taken place, the clinical symptoms abate and become more chronic, but the pulse, the temperature, and the blood-count do not become normal. Instead of being localised, the abscess may burst into the general peritoneal cavity and cause fatal peritonitis. The same result has followed unskilful handling and injudicious purgatives. This disaster raises the mortality to 77 per cent. Surgical treatment has not yet done much to lessen this mortality, for, after operation, it has been as high as 73 per cent. Or the pus may escape in other directions which are mainly dependent upon the position of the abscess. One at the ileo-caecal junction, or behind the caecum, may open into the caecum or colon, and the pus appear in the stools. This, however, occurs less seldom than is thought; for without a microscopical examination, intestinal mucus is easily mistaken for pus. The pus sometimes enters the iliacus muscle and tracks down the thigh. It has been

known to enter the psoas. The passage of pus down the iliacus is a suspicious circumstance. I have twice seen it occur in tuberculous disease of the caecum; and in other cases the recovery has been slower than usual. When the appendicular abscess is on the brim of or within the pelvis, the pus may enter the urinary bladder. But this, too, is rare; the presence of the pus is more likely to be due to pyelitis, cystitis, or to a renal calculus. The pus of an appendicular abscess has been known to enter the ureter. I have seen the pelvic abscess discharge into the rectum, and it has been known to burst into the vagina and also into the uterus. This suggests that pelvic abscesses may be opened per rectum or per vaginam. I have done both with excellent results, but only after the diseased appendix itself had been taken away. Sometimes the pus from an appendicular abscess tracks along the right colon, until it gets between the liver and diaphragm. The appendicular origin of a subphrenic abscess is to be suspected, although no signs of appendicitis are to be found. The pus, too, may penetrate into the right pleural cavity. The pus of appendicular abscesses has been known to escape into the great iliac veins. Also, I have seen it escape through the loins and simulate a lumbar abscess, and I have also seen it enter the gluteal region in a case of bilharzial appendicitis.

Differential Diagnosis.—Although ordinary acute attacks of appendicitis can be diagnosed with certainty, yet they have occasionally been confused with other diseases, even with pneumonia, the onset of which is sometimes accompanied by considerable pain, tenderness, and rigidity of the right side of the abdomen. The accompanying respiratory symptoms are, however, clearly marked. I have seen the mistake made although the sputum was already of the characteristic rusty hue. The caecal pain and tenderness which accompany enteric fever, and which may sometimes be associated with typhoid ulceration of the mucous lining of the appendix, have been a source of perplexity.

In children, when the greatly inflamed appendix or appendicular abscess rests upon the iliacus muscle, the flexion of the right hip is like that caused by an acute arthritis. Such a case calls for a skiagram and an examination under an anaesthetic. Diseases of the caecum, colon, and ileo-caecal valve sometimes cause local and general symptoms, which have been attributed to the appendix. A swelling in the right iliac fossa may be due to inflammatory exudation or suppuration caused by appendicitis; but, on the other hand, it may be tuberculous, malignant, actinomycotic, or merely a faecal accumulation. Tuberculous peritonitis may simulate appendicitis very closely, although the appendix be not infected. The tumour of an intussusception has been mistaken for an appendicular one.

The caecal pain and tenderness met with in females whose caecum and colon contain scybala ought not to mislead. Mucous and mucomembranous colitis present greater difficulties. Tenderness at the sigmoid flexure and throughout the colon, and the appearance of mucus, mucous casts, and intestinal sand after castor oil clear up the diagnosis.

The absence, too, of any marked variations in pulse and temperature assists. The patients are usually females and neurotic. The diagnosis has to be made cautiously, because chronic appendicitis may cause mucus in the stools. Light may be thrown upon such doubtful cases by ascertaining that an attack of appendicitis has been seen by a competent observer. When the mucus in the stools is caused by the presence of an inflamed appendix in the pelvis, error is easily fallen into, but the type of patient—often a young and vigorous man, the tenderness elicited by rectal examination, and the marked variations in the pulse and temperature—exceeding such as occur in mucous or muco-membranous colitis, help in the diagnosis. Twice in cases of appendicitis with colitis, the pain and tenderness were for a time upon the left side along the course of the sigmoid flexure and left colon. After some days or weeks, however, they disappeared from this region, and were found in the usual place. Mucous and muco-membranous colitis may spread to the appendix, and occasionally the appendix has been removed with a marked diminution in the pain and suffering. It is, however, to be remembered that in such circumstances a cure cannot be promised. Lead colic has been mistaken for appendicitis, and an acute attack of appendicitis during the course of lead colic has been overlooked. Sometimes appendicitis is closely simulated by cholecystitis. After a time, however, the pain and tenderness become localised at the site of the gall-bladder. Cholecystitis and appendicitis have in several instances been found to coexist. Symptoms variously attributed to cholecystitis, renal calculus, and appendicitis were found to have been caused by the last. On another occasion pain and tenderness about M'Burney's point were caused by cholelithiasis. Cautious investigation over a period of time usually clears up these ambiguous cases, but in some an accurate diagnosis can only be achieved by an exploratory incision. The pain and tenderness of renal and ureteral calculus have been mistaken for the pain and tenderness of appendicitis. The presence of lumbar pain, and of blood, pus, and crystals in the urine, should excite suspicion, and a skiagram should be taken. I have operated upon a patient in whom appendicitis and renal calculus were present at the same time. Pyonephrosis has been mistaken for appendicitis, as have perinephritic abscesses, and renal tumours. Abnormal mobility of the right kidney may cause pain like that of appendicitis, or owing to accompanying enteroptosis the appendix may have been bent or kinked. The pain and tenderness which are associated with duodenal ulcer have some resemblance to the pain and tenderness of appendicitis, but are higher up.

In females, appendicitis is not infrequently mistaken for inflammation of the right ovary, Fallopian tube, and broad ligament. The diagnosis of ovaritis on the right side should be received with scepticism. In my experience, the ovary does not easily become inflamed or suppurate. It is, as I have already said, by no means unusual for the inflammation of appendicitis to spread to the pelvic contents. I have seen the symptoms caused by a small ovarian cyst with a twisted pedicle,

and also those caused by a suppurating ovarian cyst, mistaken for appendicitis. A small appendicular abscess at the right brim of the pelvis may closely resemble a pyosalpinx. The pain, tenderness, and swelling of a haemato-salpinx or of a small haematoma on the right side resemble very closely those of appendicitis. The inflammatory symptoms which followed the rupture of a small ovarian cyst were also attributed to inflammation of the appendix. When appendicitis sets up acute peritonitis of any considerable extent, the retention of flatus and of faeces, the abdominal distension, and the vomiting are sometimes attributed to mechanical causes; but the heightened temperature, accelerated pulse, leucocytosis, abdominal tenderness, and absence of vermicular movements should help in the diagnosis. An inflamed appendix may, however, become adherent and produce a mechanical obstruction, or cause omental adhesions which have also been known to cause mechanical obstruction. A history of appendicitis is, therefore, an important clue in intestinal obstruction. In cases of acute general septic peritonitis it is sometimes difficult or impossible to tell whether the starting-point was the vermiform appendix, or the stomach, intestine, gall-bladder, or uterus and Fallopian tubes.

In acute appendicitis, rupture of an abscess into the general peritoneal cavity usually produces shock and collapse followed by intense and widespread abdominal pain and tenderness. Inflammatory obstruction supervenes. The abdomen becomes uniformly tight and distended, motionless, and very silent; quantities of thin, offensive fluid are vomited up—mouthfuls at a time. The gurglings and noises of vermicular movement are not heard with the stethoscope, but only the occasional splash of dropping liquid. The temperature is not as a rule high, but the heart beats quicker and quicker, and at last stops from exhaustion. Towards the end the tenderness and pain may cease, and from septic intoxication, the patient may become excited and at last delirious. A cold sweat covers the brows, and the hands and feet are icy cold. When the peritonitis is caused by the more gradual spread of the infective inflammation along the peritoneum, the onset is most insidious, but attention to local and general signs affords the clearest indications. The inexperienced are sometimes thrown off their guard by the mental condition of the patient. Their physical condition, too, may be extraordinary. I have known a man whose abdominal cavity contained a gangrenous appendix and quantities of pus walk about the bedroom, stand up and shave himself, and allow the abdomen to be freely handled. His pulse was rapid, and he had great rectal tenderness.

Treatment.—*Expectant and Palliative.*—The cure of appendicitis by rest, dietary, and drugs is uncertain. This is obvious in the light of some of the causes of appendicitis: concretions, foreign bodies, bends, torsion, stricture, empyema, mucocele, tuberculosis, cancer, or actinomycosis. But it is reasonable to suppose that under favourable conditions some of the other causes of appendicitis may cease to act. For instance, faecal or septic contents may be expelled into the caecum; or bends or

kinks may straighten out and allow the appendix to clear itself; or it is just conceivable that a torsion might become untwisted. Inflammation, too, of the mucosa, and especially of its lymphoid tissue and lymphatics, might reasonably be expected to subside, as it does in other parts of the body. The cure of ulceration is less likely, and the cicatrisation of ulcers may end in permanent stricture or stenosis. But no one can accurately infer the pathological conditions underlying an attack of appendicitis. In about 80 per cent the cause is ulceration of the mucosa with septic contents and bacterial invasion. Therefore, in every case, the greatest vigilance must be exercised, and the prognosis given with diffidence. It is to be remembered that chronic appendicitis may, through want of care, become acute; and acute appendicitis may end in abscess or in septic peritonitis.

The diagnosis and treatment of chronic and subacute appendicitis generally fall to the physician. Rest, a digestible diet without fruit or vegetables, and the careful avoidance of sudden changes of temperature, are the main considerations. The condition of the teeth should be looked to, and careful mastication enjoined. Ill-cooked and hasty meals are injurious. Warm woollen underclothing is of great importance. As I have already said, those who suffer from appendicular colic, or from chronic appendicitis, may and usually do have a heightened body temperature. Their smouldering appendicular inflammation easily blazes up into an acute attack. Attendance at a garden-party, a funeral, a theatrical performance, have preceded attacks, and oftentimes railway journeys and violent games, such as football. Drugs, such as salol, salicylate of soda, or β-naphthol seldom (beyond lessening the pain for a time) do good; mild aperients, such as cascara sagrada, senna, or castor oil, may be cautiously given, especially a dose of castor oil when the stools are wanted for inspection. A judicious dose of castor oil often brings to light a mucous or muco-membranous colitis. The safest way of unloading the bowels is to introduce three or four ounces of olive oil into the rectum at night, and use a soap and water enema in the morning. In gouty subjects, pain in the right iliac fossa may be cured by calomel and the usual remedies for gout. I have observed no relationship between so-called rheumatism and appendicitis. Should the pain, tenderness, discomfort, elevation of temperature, and inability to work regularly or to enjoy life continue, then appendicectomy can be performed. During the quiet stage, this operation is one of the safest of abdominal operations. The mortality is not more than 1 per cent, and in experienced hands is less. "It is probable that the mortality of perityphlitis, taking all phases of the disease together—the most trifling attacks with the most serious—is about 5 per cent" [Treves]. In the absence of acute peritoneal inflammation, the appendix is usually removed through an abdominal incision less than three inches long, and through which the fibres of the abdominal muscles are separated and not divided, so that they can be accurately brought together with silk and without any fear of ventral hernia. The appendix itself, too, is less

difficult to find, and, after its mesentery has been easily secured, can be removed with accuracy, and the small intestinal wound closed with sutures which hold securely, and prevent any chance of a faecal leak. During such an operation, the bleeding is trifling and may not exceed a tablespoonful.

Operation during the Acute Attack.—But during an acute attack the intestines are usually distended, and the inflamed peritoneum bleeds readily, so that the usual landmarks are obscured; the appendix is much more difficult to find, and in consequence more of the abdomen is disturbed; the inflamed meso-appendix is, perhaps, friable and difficult to secure; and the inflamed caecal peritoneum forms a bad holding-ground for sutures. Owing to the oozing of blood continuing, it may be necessary to drain the abdomen, and this increases the risk of ventral hernia. Should silk sutures have been used instead of catgut they may reappear, having become infected from the inflamed peritoneum which swarms with bacteria; finally, after the operation the usual complications of abdominal operations—shock, vomiting, and flatulent distension—are more severe and more uncontrollable. The peritoneal adhesions disappear in a remarkable manner. At an operation done during the quiet stage hardly any may be found although they were numerous when the abscess was opened. I have often observed that the physician has attached more importance to adhesions than the surgeon, and has therefore been more apprehensive as to the dangers and difficulties of the operation. Appendicectomy during the acute attack, and before suppuration has taken place, is attended with a higher mortality. At the London Hospital (18) 6 out of 34 died, or about 17·6 per cent. The operation, however, is done to cut short acute septic inflammation, and to prevent the onset of suppuration, which, if left alone, has a mortality of 48 per cent (Bull). Left alone, death ensues usually before the end of the eighth day. According to Fitz's table (quoted by Treves) 4 per cent die within forty-eight hours, and 22 per cent before the fifth day. The highest death-rate (24 per cent) comes between the seventh and eighth days. As yet, medicine can do little more than guide the course of septic inflammation, it cannot control it. My own experience tends to shew that it is safer to operate early upon cases which begin suddenly with very acute pain and tenderness, distension, accelerated pulse, heightened temperature, marked leucocytosis, and, perchance, a rigor. In such the acute septic inflammation may never become localised, or, if it does, the tardy evacuation of the abscess is more dangerous than the immediate operation. Some small localised abscesses when left alone incubate virulent bacteria, which, when let loose, cause violent sepsis and even gangrene of the abdominal walls. But in appendicitis of moderate severity, in which the pain, tenderness, distension, pulse-rate, and temperature all tend to subside, and especially if flatus be passed naturally and freely, procrastination is allowable, for several advantages are gained by tiding over the attack. After that has subsided, an

operation may not be required, or, if required, can be done under the most favourable local and general conditions. The expectant treatment of acute appendicitis is attended with a mortality which has been variously estimated. Fitz has put it as high as 11 per cent; Dr. West (18) at about 5 per cent; Nothnagel gives statistics of perityphlitis shewing a mortality of 10 per cent; Fürbringer 8 to 9 per cent; Reuvers about the same; and Guttmann 4 per cent. But clinical statistics are fallacious, and doubtless many who died of general peritonitis, subphrenic and hepatic abscess, portal pyaemia, mesenteric thrombosis, and so forth, have not been included, although the disease had originated in the appendix.

In treating an acute attack of appendicitis, it is to be borne in mind that the appendix may be upon the point of perforation; also that there is an acute septic inflammation of the adjacent peritoneum. The latter is comparable to an acute cellulitis and lymphangitis of the arm or leg, and the lymphatic glands are similarly swollen and inflamed. The patient should be in bed and recumbent; the muscular system and heart as still as possible. Any movement, or any digital examination, should be very gentle and very circumspect, for more than once an abscess has been ruptured. The bed is on no account to be left for defecation or urination. The room should be kept at an equable temperature of 65° F., and the surface of the body protected from draughts. It may be assumed that cold applied to the exterior of the body congests the interior, especially if it be already inflamed. In an attack of any degree of severity, a day and a night nurse are required. Changes may occur with rapidity, and therefore the pulse and temperature must be recorded every six hours. When doubt exists as to the progress of events, a blood-count should be made once at least in the twenty-four hours. An increasing leucocytosis is ominous, but too much is not to be inferred from one which is stationary or falling. It is undoubtedly safest to relieve the bowels by means of enemas given with a gravitation tube and funnel. Of the various enemas in use, soap and water thoroughly mixed with castor oil, with turpentine, or with both of these, and tincture of asafetida, are perhaps the commonest and the most efficacious. Castor oil and turpentine are remedies which are nearly always at hand. In the first enema half-ounces of each are given, in the later ones an ounce. I have seen no ill effects whatever follow when these enemas have been retained—even for several hours. Should enemas not suffice, an aperient may be required, and this calls for considerable judgment, as an operation may be a better alternative. Calomel is one of the best aperients, for it acts quickly and with a minimum of pain or colic. Two or three grains of calomel are given for the first dose, and then a grain each hour until the bowels act. The action of calomel may be helped by giving each hour a drachm or two drachms of sulphate of soda or of sulphate of magnesia.

The pain which is sometimes so acute at the beginning of an attack may be allayed by warm fomentations applied to the abdomen. The

stupes may be liberally sprinkled with laudanum. As an operation may be needed, leeches and applications which might injure the skin and render it septic should be withheld. For the further relief of pain I usually prescribe small doses of laudanum by the rectum. To adults may be given thirty minims in water, and this may be repeated in an hour if necessary. Given in this way the laudanum has a most soothing effect, and is comparatively harmless. Morphine is to be given with reluctance and in small doses ($\frac{1}{8}$ to $\frac{1}{6}$ gr.). Morphine masks the pain and tenderness, so that the extension of the peritonitis is overlooked, and a spurious feeling of relief is experienced. It also intensifies the intestinal paralysis and distension, and, as Greig Smith pointed out, the distended intestines obstruct each other, so that the majority of cases of acute septic peritonitis end with obstruction. Morphine, too, is a dangerous addition to the toxins already in the blood, some of which are like it in effect, for they produce a kind of apathy and coma.

The treatment of appendicular abscess is the evacuation of the pus at the earliest possible moment. I have had to regret delay, but not a prompt search for the matter. As I have already said, the pus of localised abscesses incubates virulent bacteria, which, when let loose, cause acute septic inflammation, and at times gangrene of the newly exposed tissues. The patient, too, seems to have become less resistant. As regards surgical interference, each case must be judged on its own merits—no two cases are exactly alike. As a rule, the question can be decided upon the lines which have been laid down. There will be little delay when the numerous dangers of delay are clearly understood.

Operative Treatment during the Quiescent Stage.—The first vomiting, due to the anaesthetic, the flatulence, the discomfort, and the backache which follow abdominal operations can be lessened or prevented altogether by careful and thorough preparation. Those cases in which the diagnosis requires verification by a probationary period in bed suffer much less than those who will not (or cannot) have a short rest and preparation. But, whenever possible, forty-eight hours should be passed in bed; the diet should be light, without fruit or vegetables, and the alimentary canal should be well cleared of its faecal and bacterial contents. The troublesome and distressing flatulence and flatulent distension which follow abdominal operations are in no small measure caused by the intestinal bacteria converting the slowly digestible foods, especially fruit and vegetables, into gases. If the patient has been in the habit of taking aperients without ill consequences, then more may be given to clear the intestines. Half an ounce of castor oil may be given for the first dose; if that does not act in four hours, then a teaspoonful may be given each hour until the bowels act. Castor oil is safe but unpleasant. If another kind of aperient have been used with success, it may be taken instead of castor oil. But not infrequently aperients cause abdominal pain, discomfort, and even vomiting, so that enemas, such as those just mentioned, have to be resorted to. No food or drink whatever should be given by the mouth for six, or, better still, for twelve hours before the operation.

It is customary to give a light meal at about 8 P.M., and nothing whatever until after the operation, done at about nine the following morning. As a rule, a warm bath is admissible the night before the operation, and the hair should be removed completely from the whole of the field of operation. It is unnecessary to cover the skin within the field of operation with unpleasant and disagreeable antiseptic compresses. They do harm rather than good. In women and children the skin within the field of operation is usually prepared during anaesthesia. If necessary, it is shaved, and then washed and scrubbed with hot water and soap; the fat is removed from it with ether or turpentine; and it is finally disinfected with the author's spirit and biniodide of mercury lotion. The technique of the operation need not be given in detail. The recumbent posture is the best, but when the appendix is adherent within the pelvis, Trendelenburg's position enables the operator to see what he is doing, and thus conduces to safety. The incision through the abdominal wall must fulfil the usual requirements of abdominal surgery: (*a*) the muscular and aponeurotic planes should be split and pulled aside, and not be cut across; (*b*) it should be planned so as to be brought together, layer by layer, with buried sutures, and to come together and not go apart during straining and vomiting. The abdominal wound is usually about three inches or less in length, and is usually closed layer by layer with interrupted sutures of twisted silk. Afterwards an occlusive dressing is applied with firm bandages, so as to prevent the entrance of air during vomiting or restlessness. The after-treatment usually consists in: (1) the prevention of shock; (2) the relief of pain; (3) the relief of anaesthetic vomiting; (4) the relief of flatulence. It is unnecessary to dwell upon these, for they are merely parts of everyday surgical practice. The dressing is removed on the eighth or ninth day, the skin sutures taken out, and a collodion dressing applied. As in other abdominal wounds, the risks of ventral hernia have to be taken into consideration, and the patient kept in bed for three weeks, and then have three weeks on a couch followed by six weeks' gentle exercise. During the second three weeks carriage exercise is permissible, and during the final six weeks a light occupation may be undertaken.

Suppuration is now rare, and in consequence ventral hernia is rare too.

Appendicular abscesses require to be opened at the earliest possible moment. The site of the incision is usually determined by the situation of the abscess, and should be planned so as to open and drain the abscess with the least disturbance of the general peritoneal cavity, and the least damage to the abdominal walls. The abdominal wound heals mainly by granulation, so that the risks of ventral hernia are considerable. When the abscess is in the iliac fossa the oblique incision is suitable; but it may be necessary to cut some of the aponeuroses and some of the muscular fibres to provide thorough drainage. An abscess in the right flank can be opened by a similar incision, but farther to the right. These flank abscesses usually require a counter-opening near the outer

edge of the erector spinae. A pelvic abscess is best opened by an incision through the outer fibres of the right rectus abdominis. No ill effect follows the division of the deep epigastric artery and veins. Or the pelvic abscess may bulge towards the median line, and then the incision is made through the inner fibres of the rectus abdominis. I have opened pelvic abscesses successfully both per rectum and per vaginam. Acute appendicular abscesses cannot always be opened without some exposure of the general peritoneal cavity; but extravasation of pus and blood can be prevented by gauze packing. When the abscess has been opened, the question of the removal of the appendix arises, and is decided according to the conditions prevailing at the time. In very acute cases in which the appendix is intensely inflamed, perforated, or gangrenous, and surrounded by thin pus, it is easily found and removed. In such circumstances the peritoneal surfaces are not firmly adherent, and do not bleed very freely. The high mortality in this class is not due to the removal of the appendix, but to the virulence of the infection. There is a second class in which the pus is thick and localised by peritoneal adhesions, and in which the haemorrhage is rather copious. In a quarter or more of these the appendix lies in an accessible position, and can be removed with hardly any additional exposure of the general peritoneal cavity, risk of extravasation of pus or blood, or of lacerating the inflamed and adherent intestines. Should it be deemed injudicious to continue the attempt because of the haemorrhage and risks of extravasation and laceration, it may afterwards, in not less than 15 per cent of cases, be necessary to remove the appendix. Appendices which operators have failed to remove have shewn concretions and septic contents, pins and other foreign bodies, ulceration of the mucous membrane and chronic sepsis, stricture near the caecal end, and intra-abdominal appendicular fistula (*vide* Fig. 24). In some of these there has been a persistent sinus or a faecal leak. Both tuberculosis and cancer of the appendix occasionally lead to the formation of abscess, and the prospects are, of course, more favourable when the tuberculous or cancerous appendix has been completely removed. Should it be necessary in ordinary cases of acute abscess to remove the appendix after the abscess is well, the operation is not as a rule of a particularly complicated nature. Peritoneal adhesions disappear in a remarkable manner. But when a septic sinus or faecal leak is left, the dangers of the operation are much greater. Finally, there is a third class, in which the abscess is large and chronic, and hemmed in by firm and tough adhesions from amidst which it is obviously impracticable to extract the appendix without lacerating the intestines, and, therefore, the attempt has to be abandoned. But in this class I have seldom known subsequent troubles arise. For operations on septic cases catgut is used for ligatures and sutures, together with reinforcing sutures of fishing gut in the abdominal wall. To overcome the intestinal distension and paralysis, usual complications of acute appendicular abscess and of septic peritonitis, an aperient, such as sulphate of magnesia, may be introduced into the

caecum through the hole at the stump of the appendix (Sheild). Or small and repeated doses of calomel should be given as soon as possible after the operation. The first dose usually consists of from three to five grains, the subsequent ones being a grain each hour. When the appendicitis is complicated by diffuse septic peritonitis, it may be desirable to empty the distended intestines by means of small, multiple incisions. Large and efficient drainage tubes are inserted in the pelvis, and possibly in the flanks. The sepsis may also be combated by the



FIG. 24.—Semi-diagrammatic. Intra-abdominal appendicular fistula. ×14. An acute abscess had been opened for drainage. Three months afterwards a fresh abscess formed beneath the scar, and after it had discharged itself the appendix was easily removed.

continuous infusion of normal saline solution into the cellular tissues (Barnard). A faecal leak may be seen during the operation, or may appear afterwards (49 in 1000—London Hospital statistics (18)). The outcome is usually favourable, and spontaneous closure is the rule, especially when the leak is caused by an acute infective inflammation or by traumatism. Should the leak refuse to heal, an examination under anaesthesia may afford evidence that it has a lining of mucous membrane, and is not likely to undergo spontaneous cure. Or a silk ligature, or suture, or foreign body may be brought to light. An operation for the closure of faecal leaks or fistulas is rather perilous, and ought not to be undertaken until due time has been allowed for the occurrence of the changes which necessarily precede the closure of the aperture. When

the leak has been large and has allowed about half the faeces to escape, a year is, in my opinion, a reasonable period. But when the leak does not close, it is to be suspected that there is an obstruction in the large intestine, such as a stricture caused by hyperplastic tuberculous inflammation, or that the aperture is the seat of tuberculosis, cancer, actinomycosis, or other chronic infective disease. An examination under anaesthesia and a microscopical investigation might render the diagnosis clear, and also shew that an enterectomy and intestinal anastomosis could be performed. As a rule aperients by the mouth make faecal leaks worse.

A very small proportion of cases without abscess have not been cured by the operation. Out of two hundred of my own cases, in which the appendix was microscopically examined, in two the appendix was normal and in three its condition was doubtful (18), and some of these derived no benefit. In several instances the operation has failed because the whole or part of the vermiform appendix had been left behind. Sir F. Treves quoted one case in which the whole appendix was left behind, and I am acquainted with another. He also mentions cases in which appendicular stumps had been left and caused renewed attacks. In one the stump was half an inch long, and in another it was three-quarters of an inch long. A portion of the appendix may be left behind when the caecum is of the foetal type and acutely inflamed. For instance, I opened an acute appendicular abscess in a boy aged three and a half years, and removed the appendix by amputating through the cone, by which it joined the caecum. The patient got well, but a small sinus remained at the centre of the scar. This was lined with mucous membrane, which contained lymphoid follicles like those of the appendix. It was found that all the appendix had not been removed, and that its open end was fast in the scar, forming an extra-peritoneal appendicular fistula. Another three-quarters of an inch of appendix was removed, and recovery ensued. In another case an acute, localised, intra-peritoneal appendicular abscess was opened by another surgeon, but the patient did not get well, and a sinus continued to discharge. At a second operation I removed an inch of vermiform appendix with an intra-abdominal appendicular fistula at its end. When the caecal peritoneum is so intensely inflamed, as it was in that case, it is, of course, not always easy to tell during the operation whether the whole appendix has been removed. But when symptoms continue after the appendix has been properly taken away, then it is to be feared that some complication may have escaped the vigilance of the physician or of the surgeon. Or a complete error in diagnosis may have been committed, and some other disease have been mistaken for appendicitis; such, for example, as a renal or ureteral calculus or cholecystitis. Affections of the colon, of the right kidney and ureter, of the gall-bladder, of the peritoneum, as, for instance, tuberculous peritonitis; or in the female, of the uterus and its appendages, are some of the commonest complications of appendicitis.

Cautious observers are seldom deceived in the diagnosis of appendicitis, and the certainty is greatest when it is the rule to submit every doubtful

or irregular case to a period of clinical observation. During this it may be necessary to make an examination under anaesthesia, or to inspect the interior of the bladder with the cystoscope, or to take an *x*-ray photograph. When, after an operation, the microscopic examination has proved that the appendicitis was tuberculous, cancerous, or actinomycotic, the ultimate outcome is not within the control of the surgeon, as it is in ordinary cases of chronic ulcerative appendicitis. The diseases which have been mistaken for appendicitis are referred to on p. 618.

It is to be remembered that surgeons are not infrequently called upon to remove the appendix as a prophylactic measure after the departure of acute attacks of inflammation. In such circumstances the microscopical sections may be normal, or shew nothing but the faintest trace of the antecedent inflammation; but nevertheless it is unreasonable to reproach the surgeon for having removed a normal appendix. Furthermore, surgeons are not infrequently called upon to operate during the quiet stage, and when they are dependent upon others for information as to what occurred during the acute attack and for a correct diagnosis. But nevertheless it remains true that appendicectomy is one of the safest and most successful of abdominal operations.

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C. B. L.

COLIC

By W. Hale White, M.D., F.R.C.P

Colic is a very old word, which originally meant a severe, sudden abdominal pain that was supposed to be due to some morbid condition of the colon. Now, for some foreign authors, it includes any sudden, sharp abdominal pain, and, therefore, it is obvious that there may be as many varieties of colic as there are abdominal or pelvic organs, or even more. The laity have given the word a still wider significance, and we find a sudden sharp pain in any part of the body called colic, for instance, a sudden pain in the head is called "head colic." Some writers have attempted to limit the use of the word according to the supposed mode of production of the pain. Thus, Traube suggests that colic should mean the pain produced by the contraction of the muscular walls of any viscus, the natural issue of which is obstructed; while other authorities would limit the word to abdominal pains which, because they have no obvious cause, are thought to be neuralgic. I believe, however, that among medical men in England the word usually means a sharp, sudden pain, having its seat either in the pelvis of the kidney, ureter, gall-bladder, biliary ducts, or intestine; hence we speak of renal, biliary, or intestinal colic; but we must remember that to say a patient is suffering from colic is not a diagnosis. For a diagnosis the cause of the pain must be discovered. Renal and biliary colic are described in other articles, and, therefore, intestinal colic only will be considered here (see also art. "Enteralgia," p. 405).

The pain usually comes on suddenly, without warning, but occasionally it is preceded by nausea, eructations, slight intra-abdominal uneasiness or borborygmi. It is nearly always referred either to the neighbourhood of the umbilicus, or rather above it over the colon; and it may radiate in various directions about the abdomen, or even beyond it. Its most important characteristic is the griping, and the patient commonly describes it as stabbing, cutting, tearing, or boring; it is often excruciating, and the sufferer tosses about, seeking relief first in one position and then in another, but in the end usually choosing one in which he can press upon the anterior abdominal wall. Thus, he may lie on his back and rub the abdomen with his hands; or he may lie on his face, pressing the abdomen against a pillow. This relief of the pain of intestinal colic by pressure is of much importance in distinguishing it from that of peritonitis, which is increased by pressure; but it must be remembered that if the colic be associated with organic disease, or great distension of the intestine, pressure may increase the pain.

Intestinal colic is paroxysmal. The paroxysm may last a few minutes, many hours, or even a couple of days; but it usually ceases as suddenly

as it began, and the end may be coincident with the escape of gas from the anus. Often an attack consists of a series of frequent paroxysms; their number, duration, severity, and the length of the interval between them are all most variable.

In a mild case of colic the pain alone attracts attention, but if it is severe other symptoms may be observed. The mere intensity of the pain may cause the patient to shriek, and even to break out into a cold clammy sweat, and then, after it has passed away, there is a feeling of weakness and faintness. The countenance is often anxious, but it rarely assumes an extreme degree of the abdominal character. Although the temperature is usually rather subnormal, it must be remembered that occasionally it is raised a degree or so, for if this be forgotten serious mistakes may be made. When the temperature is raised the pulse is proportionately quickened; but, otherwise, if the pulse be altered, it is commonly a little slowed, and its pressure and volume diminished. The abdomen is nearly always somewhat distended; and it may be possible to note the coils of intestine stretched by the gas within them, to see the peristaltic wave pass along them coincidently with the paroxysms of pain, and to observe the collapse of the intestine when, the obstruction having been overcome, the gas escapes and the pain ceases.

Very often the abdominal muscles are reflexly contracted and the abdomen becomes hard and rigid; occasionally the testicles are retracted, and Eichhorst adds that even priapism may be observed. Nausea and the vomiting of small quantities of sour fluid are by no means uncommon; and there may be a constant desire to micturate, even with an empty bladder.

It is highly probable that the pain is due to the forcible contraction of the unstriped muscle of the intestine against an obstacle. The reasons for this belief are, that the contractions may sometimes be seen through the abdominal wall, and that the pain closely resembles that produced by the contraction of the muscle in the wall of the cystic duct, or of the ureter, when there is an obstruction due to a gall-stone or a renal calculus. It will be noticed that in both these instances the characteristic sharp, griping pain is produced by the attempt to overcome an obstacle of recent formation which may be dislodged, and that we do not usually meet with this pain when neither of these conditions is fulfilled, as, for instance, in jaundice due to the pressure on the common duct by cancerous glands in the transverse fissure of the liver. So also in the intestine, it is probable that in most cases of colic the cause of the painful muscular contraction is some obstacle which has not long been there, and which there is some chance of dislodging. This view harmonises well with the fact that ordinary colic is, of itself, rarely if ever fatal.

Dr. Herbert P. Hawkins has described cases of abdominal pain which he believes to be due to spasm of the intestine without any obstacle. In one case the abdomen was opened, and a portion of both the large and small bowel could be seen contracted. He states that the large intestine is more often implicated than the small, and the caecum and sigmoid are

the most common seats of the trouble, hence the condition is often mistaken for appendicitis. It is equally common in men and women, and most frequent in emotional people between 20 and 50. Neurasthenia and hypochondriasis are associated with it. All the sufferers are constipated. The pain comes on in attacks, the duration of which may be hours or days, and the disease may last years. Sometimes the pain is hardly more than a discomfort, and although it is relieved when the bowels are opened, a sensation remains that the process is not complete. In a very few cases it is probable that abdominal pain is due to a real neuralgia of the intestinal nerves; such cases may be called enteralgia. In a case of this kind laparotomy was performed under the impression that there was strangulation by a band (Ashe).

The commonest variety of colic is that of young children, in whom the pain is recognised by their cries, by their restlessness, and the drawing up of their legs. It is due to improper feeding, and is, therefore, much commoner in hand-fed than in breast-fed babies; it may be met with in the latter, however, when they are allowed to swallow too much milk at a time. The result of the improper feeding is that gas—due perhaps to decomposition—accumulates in the intestine and distends it, but cannot be dissipated because the presence of faeces or undigested curds prevents its passage onwards. The child is often relieved by an evacuation of the bowels, or by eructations of flatus from the mouth.

The preventive treatment consists in allowing small quantities only of food at a time; and if the child be hand-fed, lime-water or fresh barley-water should be added to each ration of milk. If even then it suffer from colic the milk should be predigested, or artificial human milk should be employed. For immediate treatment during an attack of infantile colic warm fomentations should be applied to the abdomen; in a severe case an enema of six or eight ounces of warm water should be administered, and a grain of mercury and chalk night and morning by the mouth will be found useful. In milder cases a warmed flannel binder round the abdomen, and a mixture of one grain of bicarbonate of sodium with two or three minims of tincture of gentian in dill water, or peppermint water, sweetened with a little syrup, given three or four times a day, will generally afford relief. New-born children may suffer from colic due to retention of meconium. Forgetfulness that Henoch's purpura (see Vol. V.) causes severe abdominal colic has more than once led to errors.

In adults the most important, if not the commonest, form of colic is that due to lead. This metal causes constipation, and the muscular contractions which ensue in the attempts to overcome this are particularly prone to give rise to severe colic (*vide* art. "Lead Poisoning," Vol. II. Part I. p. 1044). It is stated, especially by German authors, that copper will produce a form of colic very closely resembling that due to lead, but it is, I believe, very rare in this country.

The simplest variety of colic seen in adults is that arising from constipation; and it is obvious that this form is analogous to the biliary or

renal colic produced by gall-stones or renal calculi. Its treatment is discussed under the headings of constipation and faecal impaction.

The most characteristic colic, however, is that which is associated with certain varieties of indigestible and irritating food. Unripe hard fruits, such as apples and pears, or nuts or shell-fish in large quantities, are very common causes of it; but individual digestions differ so widely that no definite list of dangerous articles of food can be given. In these cases the colic is usually associated with constipation, and the cause of the pain is the same as in the former varieties; sometimes the irritant matter causes diarrhoea, yet even then, although the indigestible food be sufficiently irritating to set up some enteritis and consequent diarrhoea, the mass may remain, blocking up the calibre of the intestine and causing colic. The treatment is obvious; namely, rest in bed, hot fomentations to the abdomen, and a simple purge, such as castor oil, to get rid of the irritant; when the pain is severe ten or fifteen drops of laudanum may be added to it, and, should the bowels not act in a few hours, the medicine ought to be repeated. If, owing to nausea or vomiting, or for any other reason, it is inadvisable to give a purge by the mouth, a pint of warm soap and water, with some castor oil added to it, may be thrown into the rectum.

If no treatment were adopted the contractions of the intestine, after causing much pain, would nearly always overcome the difficulty in the end; the irritant would be passed and the patient would recover. The cases in which this result would not follow are those of impaction of faeces, or of foreign bodies, such as gall-stones, in the intestine; in these cases the patient is said to suffer rather from intestinal obstruction than from mere colic.

There is perhaps no ailment in which a **diagnosis** is more important than in colic. Many a patient who has died of peritonitis, acute intestinal obstruction, or appendicitis, might have been saved had he not been treated at first under the impression that he was suffering from colic due to irritating food; and although, as Fagge says, severe colic forms an exception to Sir S. Wilks's aphorism that if the pain in the abdomen is so severe as to cause the patient to send for a medical man, this in itself proves the administration of a purgative to be unjustifiable, yet the very fact that colic is the only exception should make us very cautious in our diagnosis.

Acute intestinal obstruction presents the greatest difficulty, and if the sufferer be seen early a diagnosis is often quite impossible. To such a patient a purge is nearly always administered, especially if there be any evidence that an unusual article of food has been eaten; but if this fail to benefit him, and particularly if, as testified by frequent vomiting, by his appearance, and by the state of his pulse, he obviously and quickly becomes very ill, the purgative should on no account be repeated, for the case is probably not one of simple colic; and if it is not, purgatives are the worst possible drugs to use.

Perforation of a hollow viscus, usually the stomach, is also at first sight

often regarded as colic; but it quickly either resembles intestinal obstruction or leads to obvious acute peritonitis. This accident is distinguished from colic by the fact that pressure increases the pain, the pain is more diffuse, the abdomen is not so often rigid, it does not move on respiration, the temperature is commonly a little raised, the pulse and expression are more characteristic of abdominal disease, and the vomiting is more severe. But as any of these symptoms may accompany colic, much caution is necessary.

Appendicitis is sometimes mistaken for colic, but in this malady a little care will usually prevent a mistake.

There are other conditions, which may be confounded with intestinal colic, in which an error is of less importance, for a purgative will do no great harm; it must be remembered, however, that apart from lead poisoning, severe intestinal colic is in adults a much rarer condition than is commonly supposed. In numbers of cases of renal colic the real cause of the pain is overlooked because it closely resembles that of intestinal colic, which, again, may likewise be closely simulated by that due to a stone in the cystic duct. If the stomach be distended with gas which it cannot eject, the pylorus and cardiac opening being presumably closed, the pain produced may be very like that of intestinal colic. I have also known that form of gastric pain which comes on some hours after food, and is relieved by an alkali, to simulate colic. In exceptional cases other gastric pains may give rise to difficulty; and the possibility of neuralgia of the dorso-lumbar nerves, of disease of the abdominal muscles, of spinal disease or of locomotor ataxy, must be borne in mind.

There are many conditions of the intestine which induce pain very like the colic just described; some authors would say that such conditions give rise to colic, others that the pain was colicky. It matters not which word is used, so long as it is remembered that organic disease of the intestine may be confounded with the class of cases I have been describing. For instance, in enteritis, simple non-ulcerative colitis, mucous colitis, ulcerative colitis, and intestinal malignant disease, the pain may be sudden and griping, and thus give rise to misapprehension. *Ascaris lumbricoides*, by reflex action, occasionally sets up paroxysmal painful attacks; and, lastly, griping pain commonly results from purgatives. Some authors describe a rheumatic colic, and suppose that such cases are due to exposure to cold; but we have very little exact information about them, and they are certainly far from common. Ingestion of cold liquids or ices will occasionally give rise to colicky pains. It must not be forgotten that certain cases of influenza are ushered in with sudden pain in the abdomen.

We now pass on to a very difficult subject; namely, to consider how far some cases of colic may be called functional and fall under the heading of neuralgia or hysteria. It must be borne in mind that the sensitiveness of the intestinal mucous membrane varies very much in different persons; and, as I have said, some articles of food, which to a strong, healthy man would be innocuous, might in a weak, sickly woman act as irritants and

give rise to colic. We must also remember that the abdominal organs are much under the influence of nervous conditions. For instance, hysterical vomiting is not uncommon. Numbers of kidneys are exposed by surgeons under the impression that a stone is present and none is found; and in one case, the operation having proved fatal, no stone could be discovered at the post-mortem examination. Fright will produce diarrhoea, and lunatics often complain of painful intra-abdominal sensations. There is then a considerable probability that some cases of colic may fall under the heading of neuralgia or hysteria; but it must never be forgotten that such a diagnosis should not be final until every attempt has been made to exclude it (*vide* p. 406). The most troublesome hysterical cases are those with intense pain, rigidity of the abdominal muscles, and superficial cutaneous tenderness. In these cases the resemblance to peritonitis is very close, but it will usually be found that deep pressure is not more painful than superficial, that pressure is not objected to if the patient's attention is distracted, and that other hysterical symptoms are present.

In nervous women pain closely resembling colic may be due to disease of the pelvic organs.

Many authors have suggested that in cases of colic organic disease of the intra-abdominal ganglia and sympathetic nerves might exist; there is, however, no evidence of this. I have had patients under my care in whom the abdominal ganglia were invaded by growth, but they had had no colic. I have elsewhere (3) shewn that in the human adult the semilunar ganglia are extremely variable, both macroscopically and microscopically. Of the morbid anatomy of the intra-abdominal sympathetic fibres we know nothing.

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W. H. W.

CONSTIPATION

By Sir Lauder Brunton, M.D., F.R.S.

Physiology of Faecal Evacuation.—In order to understand clearly the action of the various conditions which produce constipation and diarrhoea, it is necessary to consider the physiology of the faecal evacuation. The intestinal contents are moved onwards by peristaltic action in a direction from the stomach towards the anus, until they arrive at the rectum, where

their presence, by reflex action, calls upon the muscles of the abdominal wall to aid in their expulsion.

The movements of the intestine are of two kinds: (i.) peristaltic, in which contraction takes place at some part of the bowel, and gradually passes down for a certain distance; (ii.) swinging movements.

It is very rare for peristaltic movement to continue along the whole bowel; it usually extends for a certain distance only, and then stops; but a new movement may begin shortly afterwards at the place where the first peristaltic action stopped, so that by successive movements of different parts of the bowel the contents are moved on from the one end to the other. The swinging or pendulum-like movements are probably produced chiefly by the longitudinal fibres. When observed in the intestines after the abdomen has been opened, they do not seem to have any particular effect one way or another; but in all probability they are useful in restoring the bowels to their former position after they have shifted from it by peristaltic action.

Peristaltic action is usually moderate; but it may sometimes be exceedingly violent, both in amount and duration. Occasionally, violent peristaltic action occurring over short distances, but not extending down the bowel, appears simply to give rise to borborygmi; but, when excessive, and extending along the whole length of the intestine, it gives rise to diarrhoea. The ordinary peristaltic action and the swinging movements may be regarded as the normal movements of the bowel. The excessively violent peristaltic action which gives rise to diarrhoea may be looked upon as pathological. A common form of pathological movement is a tonic contraction, which converts the soft and elastic bowel almost into a rigid rod. Not infrequently this can be felt through the abdominal wall in persons suffering from chronic intestinal catarrh, from obstruction, or from abnormality in the nerve-supply, as in lead colic; and when it occurs extensively, as in lead colic or certain diseases or abnormal conditions of the brain, the abdomen becomes retracted, and assumes the form to which the name "scaphoid" has been applied. The short peristaltic actions which give rise to borborygmi may be looked upon as intermediate between pathological and physiological conditions, inasmuch as they do not occur in ordinary circumstances, but yet cause no pain or other abnormal symptom. They are so commonly due to emotional causes that their occurrence was formerly regarded as an evidence of the emotion of compassion; and, indeed, the term "soundings of the bowels" was used by the old Hebrew prophets, for example, as a synonym for compassion itself: "Where are the soundings of thy bowels towards me?" or again, "My bowels shall sound like a harp for Moab."

It has been supposed by some authors that in peristaltic action the stimulus to contraction is simply passed on from one muscular fibre to another; but this seems unlikely, and it is much more probable that, although the stimulus may travel to some extent along the muscles, it does so chiefly along the nerves. The movements of the bowels originate in nervous impulses supplied to the muscular fibres by Auerbach's plexus,

which lies between the longitudinal and circular layers of the intestinal muscle. The small intestine, and the greater part of the large, have their movements regulated from the spinal cord and sympathetic plexuses. As the greater part of the intestine works independently of the will, the nerve fibres which reach it from the cord pass through the gangliated chain of the sympathetic as well as through plexuses in the abdomen itself. The stomach and rectum, however, are more under the control of the will, and they are partly innervated directly from the brain and spinal cord. The stomach receives its innervation in part directly from the brain through the left vagus, and in part indirectly through the right vagus which enters the solar plexuses. The lower part of the sigmoid flexure and the rectum receive fibres from the first, second, and third sacral nerves, which do not pass through the gangliated sympathetic cord, but enter into the hypogastric plexus and then pass onwards to the intestine. The secretion of the small intestine has its regulating centres in the superior and inferior mesenteric plexuses; for if all other parts of the nervous system be destroyed while these are left intact, no abnormal secretion occurs; but if they are destroyed a copious secretion takes place in the small intestine, just as if all the nerves which pass through it had been cut.

The vagus and the splanchnic nerves which enter into the solar plexus have different functions. The vagus, as a rule, stimulates intestinal movements, while irritation of the splanchnics arrests them; but, under certain conditions, these effects may be reversed, and the vagus may have an inhibitory and the splanchnic a stimulating action. It has been stated by von Basch that the vagus innervates the longitudinal muscles of the intestine, and the splanchnic the circular muscles. The splanchnics also contain vasomotor fibres which cause the intestinal vessels to contract, and, through the anaemia which they can thus produce, may exert a secondary action upon the intestine. Both the vagi and the splanchnics are afferent as well as efferent nerves; for whereas the vagi probably may convey a sensation of pain, it is certain that the splanchnics do so when strongly stimulated.

From the description just given of the nervous connexions of the intestine, and of the different results which may be produced by stimulation of the nerves under different conditions, it is obvious that stimulation of the mucous membrane, either mechanically or chemically, may give very different results at different times. At one time it may cause increased secretion, at another dryness of the bowel; at one time increased peristalsis with diarrhoea, at another diminished peristalsis and constipation. A moderate amount of distension, either by faeces or gas, usually increases the peristaltic movements; excessive distension, on the other hand, tends to their abolition.

The emotion of compassion, as already said, usually tends to produce limited peristalsis with borborygmi; the emotion of fear to cause general peristalsis with evacuation of the bowels. In some persons emotions of almost any kind, even pleasurable, have a similar action; but depressing

emotions, such as anxiety and grief, frequently appear to cause not only dryness of the tongue, but also dryness of the intestine, sluggish peristaltic movement, and great constipation. Excessive stimulation, either of the inhibitory or motor nerves of the intestine, appears to result in a reaction, during which fibres of the opposite kind have the preponderance; so that diarrhoea is apt to be followed by constipation, and conversely. This result may ensue whether the diarrhoea or constipation be caused by the administration of purgatives and astringents, or be brought about by wholly different causes. It is evident, also, that if the opposing nerves, either for secretion or for motion, should be nearly exactly balanced in their excitability and power, no irritant applied to the intestine would have any result, however strong it might be; and consequently most powerful purgatives, such as jalap, elaterium, or croton oil, might be given without producing purgation.

Constipation is a tendency to the retention of faecal matters in the intestine; the evacuations either being too small or occurring too seldom. Constipation is one of the commonest troubles of civilised life; and it occurs so frequently in people who are otherwise perfectly healthy, that to a great extent it must be regarded not as a disease, but as the natural result of artificial conditions acting upon a healthy body. The natural stimulus to the movement of the bowels is afforded by the mechanical or chemical irritation exerted by the indigestible residues of food. In savage life these are abundant, because the food is not only coarse in quality, but imperfectly cooked. In civilised life the indigestible parts are to a great extent removed from food; and even those that remain are so much softened by cooking that they do not exert the same irritating action upon the bowel that they would otherwise do. Thus, in primitive communities the grain of all kinds, which forms such an important element in food, is bruised or pounded; and the meal, with a liberal admixture of sand and dust, is made into cakes, or boiled without further preparation. But in highly civilised societies the case is very different, for a great deal of the outer part of the wheat or other grain is removed, and the fine starch contained in the interior is the only part employed in making bread. This fine starch is wholly digested in the intestine, leaving little or no residue to stimulate the bowels; whereas the exterior of the grain, consisting as it does of hard cellulose, is almost indigestible, and passes through the whole intestine with very little change. It frequently happens that when people no longer restrict themselves to bread made with fine flour, and return to the more primitive bread, made either from whole meal or from bran, the bowels which had previously been constipated became regular. Many of the dietetic methods of treating constipation consist in giving food which contains cellulose in a more or less hard and indigestible form. Thus, patients are encouraged to eat bread made of whole meal or bran; apples stewed or raw; figs raw or in puddings; prunes or marmalade. Marmalade partly consists of the hard skin of the orange, which is cut into strips, and being very sparingly digestible exerts a mechanical stimulating effect upon the

bowels; in addition to the mechanical effect, however, there is a physical, perhaps we might also say a chemical action exerted by sugars and neutral salts of the alkalis. Both of these are constituents of fruit, and the neutral salts without the sugar occur in other vegetables. The sugar exists in large quantities in such fruits as figs, prunes, tamarinds, and the like; and sugar, given alone in the form of honey, golden syrup, or still better, the old-fashioned black treacle, tends to act as a laxative. It is, perhaps, still more efficient when given as a marmalade or jam, because the action of the sugar is combined in these substances with the mechanical stimulation contained in the skins or seeds of the fruit. Vegetables, such as cabbage, spinach, sprouts, and salad, have a double stimulant action, in part mechanical from their indigestible constituents, and in part chemical from the neutral salts they contain. Moreover, some of them increase the movements of the bowels by giving rise to the evolution of a certain quantity of gas which aids their expulsive action.

The causes of constipation are very numerous, but they may be ranged under two main heads: first, lessened expulsive power; secondly, increased resistance. The diminished expulsive power may be (i.) in the peristaltic action of the intestine; (ii.) in the action of the diaphragm and abdominal muscles which aid the intestine in the expulsion of its contents.

The diminution of expulsive power in the intestine itself is rarely due to alteration in the muscular fibres. In some cases it would appear that the muscular coat of the intestine is naturally weak, although the voluntary muscles in the same person may be well developed; in some men, likewise, the heart is small and out of proportion to the size of the body. The intestinal muscles, again, like the heart, may be weakened or degenerated by prolonged disease, as by fevers; but the intestines seem to have a greater resisting power than the heart to a high temperature, and do not shew degeneration to the same extent as a consequence of prolonged febrile disease. Even in severe and prolonged diseases, when the voluntary muscles have become extremely atrophied, the intestinal muscular walls are little altered. Lessening of the expulsive power of the intestine is, therefore, chiefly due to alteration in its nervous supply. Occasionally this may be of central origin, as in the extremely obstinate constipation of melancholia, mania, and other cerebral diseases; but it frequently appears to be reflex, or due to some peripheral condition in the intestinal nerves themselves. In chronic catarrh of the intestine there is a tendency to constipation, which may be due to weakening of the muscular fibres of the intestine, as well as to the nerves; in peritonitis it is probable that both agents are affected. As a rule, however, sluggishness of the intestinal movements depends rather upon the want of stimulation or the inactivity of the nervous system than upon the condition of the muscular fibres. One powerful cause of constipation is undoubtedly the presence of lime salts in drinking-water. The mode of operation of these salts has not been ascertained, but it is quite possible that it is due to an action of the lime

upon the muscular fibre, rendering its contraction slower than before; without doubt lime has this action upon involuntary muscular fibre generally.

Diminution in the expulsive power of the abdominal muscles may be due to great distension, such as occurs during pregnancy, and as a consequence of repeated pregnancies; to great accumulation of omental fat; or to tumours in the abdomen, such as large ovarian cysts. Weakness of these muscles may also be due to the innutrition consequent upon insufficient exercise. This may occur, for instance, in heart disease, when the patient is prevented by the state of his circulation from taking exercise; or it may be due to sedentary habits, or due to lack of time, opportunity, or disposition to take exercise. Again, the muscles forming the floor of the pelvis may be lax and weak, and may offer too little of the resistance which turns the faecal mass from the direction in which it passes into the pelvis forwards to the anus. There may be inability also to bring the muscles of the abdomen into full play on account of cardiac disease, which prevents a patient from straining, or from piles, fissure of the anus, fistula, or other disease in the pelvic organs; in which cases pain during defecation makes the patient afraid to bear down.

The natural stimulants to the intestines may be either of a mechanical or a chemical nature. The mechanical stimulants are the solid residues of food, or the distension by gases inside the intestine; while those from without are the mechanical pressure to which the intestines are subjected by the diaphragm and abdominal walls during the actions of respiration, locomotion, and exercise of various sorts. The chemical stimuli are certain gases—such as carbonic acid, sulphuretted hydrogen, and marsh gas, sugars, neutral salts, and possibly other substances formed by the decomposition of food by bacteria within the intestines. When the amount of food which enters the intestine from the stomach is small in quantity, or very soft and easily digestible, it naturally exercises a much less powerful stimulus upon the intestine than if it were abundant and contained a good deal of hard residue. The food may be scanty either from famine, from lack of appetite, from irritability of the stomach causing vomiting, or from ulceration and contraction of the pylorus delaying the food in the stomach. The food may be soft and readily digestible in the poorer classes, for whom bread, tea, and farinaceous food with a little sugar are the chief articles of diet; or, in the richer, from the tenderness of the food and good cooking. When the food is not only soft, but contains no indigestible materials, there will be very little residue in the large intestine; and there will be, therefore, very little stimulus to its peristaltic action. When food is too bulky and too indigestible, it is apt at first to give rise to diarrhoea, which is not unlikely to be followed by a reaction; so that those who are living constantly upon poor and sparingly digestible food may suffer from constipation as well as those in whom it is too soft or too well cooked. A similar reaction follows the use of purgatives; so that those who are accustomed to use purgatives in excess of what is required by nature are very apt to suffer from con-

stipation. One very common cause of constipation is the nervous torpor due to habit. Habits in most persons are very easily acquired. One person, if awakened at an early hour on one morning to catch a train, will go on waking needlessly at the same hour for many mornings to come; another person who, disregarding the summons to rise, once deliberately turns over and goes to sleep after his alarm has sounded, is very apt to do so involuntarily and to oversleep himself on subsequent mornings; although the alarm may sound as usual. Thus, the nervous system may be trained to excite an action of the bowels at a certain time, so that an evacuation becomes regular; but habitual disregard of the natural call to evacuate soon leads to a want of sensitiveness to it, and to constipation as the result. Some time ago I heard of a useful plan employed by a schoolmaster. He sent all the boys under his care in batches to the closets immediately after breakfast, each batch having to keep time to the minute. If any boy failed to have an evacuation in the time allowed, he was bound to report himself to the master in order to allow of proper means being employed to relax the bowels. If this plan were followed at all schools a useful habit would be established, and in many cases a good deal of discomfort in after-life might be prevented.

Prof. Cash has shewn that the introduction of food into the stomach acts as a stimulant to movements of the bowels, and the natural time for evacuation in most people seems to be after breakfast. The stimulation of the food in the morning after the long rest during the night leads to the passage into the rectum of the faecal matter which has already accumulated in the sigmoid flexure; and thus a desire to defecate is set up. Such calls are not infrequently disregarded, especially by women, from a misplaced sense of modesty; and not infrequently by men who, in their haste to business, either do not go to the closet at all, or cut the act too short. In some cases long railway journeys, or other incidents, may interfere with the daily evacuation; but whatever the cause, the result is apt to be the same. Preoccupation of mind by thought or emotion may also interfere with evacuation; and, as I have already said, cases of nervous disturbance, such as melancholia or neurasthenia, may be associated with most obstinate constipation. The natural evacuation may also be arrested, sometimes voluntarily, sometimes involuntarily, by the dread of pain or by actual pain from piles or fissure of the anus, or by inflammation in or around the ovary or some other pelvic organ. It is stated that venous stasis, especially in the portal district, is liable to cause constipation; and that, consequently, cases of cardiac or hepatic disease, which lead to portal stasis, are apt to interfere with evacuation of the bowels. This, however, is by no means invariable, and much venous congestion of the bowels may occur without the peristaltic action being lessened. It is, therefore, probable that constipation due to these causes is not directly due to venous congestion of the bowel itself, but rather to some other factor associated with it.

Increased resistance may occur (*a*) from the altered condition of the faeces—dryness, hardness, or bulkiness; (*b*) from resistance to the move-

ment of the intestine from adhesions due to old peritonitis; (*c*) to diminution in its lumen by fibrous bands passing over it, stricture of the intestine itself or of the anal orifice due to old scars, to piles, or to malignant disease; (*d*) to pressure by other parts, such as a retroverted uterus, fibroid or other tumours of the uterus, enlarged prostate or new growths in the pelvic walls, uterus, bladder, or prostate. In some cases a lessening of the lumen of the upper part of the rectum may occur from a tendency of the sigmoid to prolapse into it; though this condition more frequently gives rise to diarrhœa (*vide* p. 665).

Increased resistance to the passage of the faecal mass along the intestinal canal may arise from dryness of the faeces. There are many causes which may give rise to this defect. Too little liquid may be habitually drunk; either because the patient is not thirsty, or habitually takes little liquid in order to avoid the necessity of evacuating the bladder in circumstances in which it might be inconvenient. Others, again, instead of taking water, take wine; and thus, to a certain extent, they cheat the natural desire for fluid. Excessive excretion by the kidneys may lead to dryness of the faeces and constipation; as, for example, in cases of diabetes, where the sugar acts as a diuretic; or where food is taken containing a sufficient amount of neutral salts to act as a diuretic, but not enough to stimulate the intestine. Much exercise, leading to profuse perspiration, may also draw off water through the skin, and dry the contents of the bowels. If the faecal masses remain in the intestine they tend to become drier and drier from absorption of the liquid they contain; so that if a person, otherwise in health, voluntarily disregards the call to defecate, and puts off the act for twenty-four hours longer, his motions are usually more scanty, darker, drier, and harder than they would have been if evacuated at the proper time. These hard, dry masses accumulate and tend to obstruct the bowel, and thus further increase the difficulty of evacuation. Thus, if a healthy man is obliged to pass over a day without evacuating the bowels, the movement on the succeeding day is likely to be not only drier and harder than usual, but frequently more scanty. At first this seems extraordinary, because one would imagine that the accumulations of two days would be much more bulky; but the absorption being greater there is less instead of more residue to evacuate. If a longer time passes without evacuation, the hardened faecal masses become pressed against one another, and the motions which accumulate in the intestine become very bulky and very hard.

Milk sometimes gives rise to exceedingly hard motions. When taken in small draughts at a time, and especially when broken up by admixture with an alkaline water, or by taking a little piece of biscuit between each sip, the milk usually falls in coagula in the stomach, which are quickly digested; but if the milk be drunk in large draughts, it may form large firm curds which, instead of being properly digested, form stringy masses of casein: these become so felted together as to form an exceedingly hard mass which is very difficult to evacuate.

Another cause of dry and hardened faeces is peculiarity in the quality

of the water drunk (*vide* p. 639). People living on a chalky soil and drinking hard water are very apt, as I have said, to suffer from constipation. Where this is the case, the patient should drink distilled water or some of the ordinary bottled waters, such as soda water, potash water, or other aerated water. Not only so, but their tea should also be made with these waters, instead of with the ordinary chalky water; as the quantity of lime in the water taken as tea is sufficient, in some people, if the water be very chalky, to keep up the constipation. It is very difficult for patients supplied with hard water to get their food cooked with water free from lime; it is better, therefore, if possible, to secure soft water for cooking purposes.

Exercise has much to do with the movements of the bowels. Want of exercise tends to cause constipation; partly, I believe, because the bowels do not get a mechanical stimulant applied to them by the contraction of the abdominal muscles. The kind of exercise taken, however, is of no little importance. In ordinary walking on the flat the bowels get little or no mechanical stimulus, because almost the whole of the exertion is thrown upon the muscles of the leg and back; but on walking up a hill, and in many of the so-called Swedish movements, the intestines undergo an alternate compression and relaxation which stimulates them powerfully. In walking uphill, for example, the intestines are squeezed between the diaphragm and the abdominal muscles, both of which parts are called into unusual action by that kind of exercise.

To one class of patients I think exercise is disadvantageous. Not infrequently delicate women, especially those who have some sort of ovarian or uterine irritation, complain that the more exercise they take, particularly walking exercise, the more obstinate the constipation becomes: whereas when they lie quiet they are perfectly well. In such cases, I believe there is a reflex inhibition of the peristaltic movements of the bowels from the irritated ovaries or uterus; and some of these patients find a strange remedy in opium. My attention was first directed to this means by the case of a woman who was under the care of my friend, Dr. Litteljohn. The bowels were obstinate, and Dr. Litteljohn prescribed half a grain of opium to lessen the pain of an inflamed ovary. To his astonishment after the opium she got a natural evacuation, which she had not had for a long time. In this patient the action of the bowels clearly appeared to be inhibited by the reflex irritation of the splanchnics, or by irritation of the ovarian nerves; and in other cases I have little doubt that a similar inhibition of the intestinal movements takes place from ovarian irritation due to walking. When the patient is at rest, on the contrary, the irritation is prevented, the inhibition of the intestine ceases, and natural movement occurs. In the same way inhibition of the intestines may occur from piles, fissure, fistula, or other disease of the pelvic organs; not only may the voluntary expulsive efforts of the patients be diminished by fear of the pain which the passage of a motion may cause, but also the movements of the intestine itself may actually be lessened.

Symptoms.—The distinctive sign of constipation is that the evacua-

tions occur too seldom, or are too scanty. The amount of evacuation daily passed by a healthy person depends very much on the quantity and quality of the food; but ordinarily the amount passed, on a mixed diet, forms a brown cylindrical mass about five to eight inches long and an inch to an inch and a half in diameter. In constipation the amount may not be half of this; and consists of small dry, hard lumps, sometimes quite round, and of the size of hazel-nuts or walnuts. In some persons the omission of a motion, or its absence even for several days together, seems to cause no symptoms, either local or general; but usually in a healthy person, if the desire to defecate be checked, the impulse to evacuate the bowel may indeed soon pass off, but it is frequently succeeded by a feeling of discomfort in the abdomen, and a sense of heaviness and dulness in the head which may amount to an actual headache. If the bowels still be not evacuated these symptoms may pass off, and remain absent for a day or two; but when the bowels continue constipated the hardened faeces may act as a mechanical stimulus to the bowel and cause diarrhoea, which brings relief. Or, instead of causing a single attack of diarrhoea, the scybala may remain and give rise to catarrh of the colon, shewing itself in alternate constipation and diarrhoea; or they may produce the symptoms usually associated with gastro-intestinal catarrh, namely, loss of appetite, furred tongue, disagreeable smell of the breath, with colicky pains, yellowness of the conjunctiva, or, if the duodenum be much implicated, even actual jaundice. Along with these symptoms referable to the gastro-intestinal canal, may be associated dulness of intellect, irritability of temper, and depression of spirits with a tendency to headache. Continuous and obstinate constipation has been charged with causing still more serious cerebral disturbance, such as giddiness, hypochondriasis, and even melancholia or mania, symptoms which have disappeared on a proper relief of the bowels. Palpitation may also result from constipation, but probably this affection is not due directly to the faecal matters in the intestine, but rather to the flatulent distension to which accumulation of faeces gives rise. The local symptoms will depend on the site and size of the accumulation. If present in the caecum or ascending colon, it will give rise to pain and tenderness, and may lead to typhlitis with consequent peritonitis. When it occurs in the sigmoid it will press upon the pelvic viscera, and by pressure on the bladder may bring about frequency of micturition and nocturnal enuresis; by pressing upon the uterus it may give rise to dragging pain and disturbance of the menstrual functions, dysmenorrhoea, amenorrhoea, or menorrhagia; by pressing on the seminal vesicles it may cause nocturnal emissions. The so-called seminal emissions occurring during defecation are generally due in part to the mechanical pressure of scybala, and in part to the increased straining which is necessary to evacuate them. When faecal accumulation is very great it may give rise to complete obstruction of the intestine and eventually to death.

Treatment.—*Habit.*—One of the most important factors in the treatment of habitual constipation is the acquisition of the habit of regularly

evacuating the bowels at the same time every day. In order that this habit should be formed the patient should go to the closet every day at the same hour, and remain there soliciting the action of the bowels for many minutes; and he should do so, if possible, by the clock with perfect precision, whether he anticipate an evacuation or not.

Time.—The time chosen should be such that it is not likely to be interfered with by avocations. In most cases the best time is the morning just after breakfast; but if the patient is likely to be unable to keep to a definite hour in the morning, it is better to fix the evening just before going to bed. The evening should also be chosen for evacuation by those who suffer much from piles or tendency to prolapse; because if the bowels are open in the morning the piles are apt to become full and congested, or the prolapsed bowel to come down and give discomfort and trouble to the patient during the whole day. On the other hand, if the bowels are evacuated just before going to bed, the parts can be washed, the piles or prolapse may be returned, and, the recumbent position being then kept for many hours, little or no discomfort will be felt by the patient. Thus the natural tendency to recovery from these diseases will be aided. While at stool, it is sometimes advisable to aid the passage of the faeces from the sigmoid into the rectum by pressing the abdomen with the ball of the left thumb over the course of the bowel from the left margin of the ribs, between the umbilicus and iliac crest, down towards the pubes. If, after a few attempts, no desire be felt, one or two drachms of glycerin may be injected into the bowel, so as to give the local stimulation to its action which faecal matter present in the rectum would usually exert. The stimulation thus afforded is frequently sufficient to start the peristaltic action of the bowel, and to bring the faeces from the sigmoid into the rectum; as well as to call into play by reflex action the abdominal muscles which usually aid an act of defecation.

Position.—The practice adopted in this country of sitting on a closet is unfavourable to the evacuation of the bowels. If the individual sit upright the nates may be pressed together, and thus afford a mechanical impediment to the exit of the faeces. Many persons avoid this difficulty by bending the body forward at an acute angle. This position has the further advantage of putting the floor of the pelvis on the strain, so that it aids in turning the faecal masses forwards out of the rectum through the anus. Such stretching is more especially necessary in persons whose abdominal muscles are flabby and lax; especially in women who have borne several children. The crouching position in which defecation naturally takes place in the open air, and in most countries, may be partially imitated in a water-closet by having a high footstool; but when there is much difficulty in evacuating the bowels it is still better to let the patient sit over an ordinary chamber-pot on the floor. In cases in which the uterus is retroverted or enlarged this position has the further advantage of allowing this organ to fall forward, so that its backward pressure may not interfere with the passage of faeces through the rectum.

Some persons find that they can evacuate most easily in the standin position with the body bent slightly forwards and the hands resting o the knees.

Diet.—Dietetic treatment is of great importance in constipation Oatmeal porridge for breakfast is a good laxative, and its action is sti further increased if golden syrup or treacle be taken with it instead o milk. Various kinds of bread have been specially recommended fo constipation, the essential point in each of them being that the flou is not so finely ground, nor the starchy particles of it so completel separated from the harder outside of the grain as in fine white flour Amongst the various kinds used are varieties of whole-meal bread, bra biscuits, and admixtures of whole meal and bran in different proportion Golden syrup and treacle still retain their laxative properties whe mixed with flour, so that various kinds of ginger-bread have a laxativ action.

All vegetables, or fruit, containing much cellulose, especially in th form of hard fibres, leave a good deal of indigestible residue in th intestine; hence they have a somewhat aperient action. Green vegetable of various kinds—cabbage, spinach, Brussels sprouts, asparagus, Portug onions, carrots, haricots, and turnips—are all useful; and some of them especially cabbage, tend to act as laxatives not only by the mechanica effect upon the intestine, but also by forming a good deal of gas in th intestine. This gas is occasionally so voluminous as to become objection able and to interfere with the use of the vegetables. Besides thes vegetables, which are usually eaten cooked, there are some, such a tomatoes, which are useful both cooked and raw. A tomato eaten a breakfast will sometimes keep the bowels regular; and a raw apple eate in the morning before breakfast has a similar effect. Stewed fruits gooseberries, apricots, apples, pears, plums, and especially prunes, hav all a laxative action, and may be used at meal-times for this purpose Baked apples and stewed prunes are most commonly preferred. The probably owe their action not only to the cellulose that they contain but to the sugar and neutral salts which are present in them. Jams an jellies made from fruit also have a laxative action. In jams there i the mechanical effect of the seeds or skins which they contain; in jellie this is absent, but the sugar and the vegetable salts render them slightl laxative, though not so much so as jams or marmalade. Syrup an honey have also a laxative action; but one of the best substances o this kind is marmalade.

Drinks.—The amount and kind of water drunk is also of grea importance. Many people drink too little water, and frequently tumblerful of cold water on rising in the morning, or on going to be at night, or on both occasions, suffices to keep the bowels open. I winter, and with delicate persons in whom the cold water tends to li heavy at the stomach, a tumblerful of hot water sipped on rising i the morning and on going to bed at night is better than cold. To th constipating effects of hard water I have referred more than once, an

people who live on a chalky soil do well to remove the lime from the water, as far as possible, by boiling it first, and after it has settled, or has been filtered, exposing it to the air so that it may regain its freshness; or it may be aerated in a gasogene. If the water, instead of carbonate of lime which is removed by boiling, contain sulphate of lime which remains in solution notwithstanding the boiling, so that the water is permanently hard, patients who are troubled with constipation should drink aerated water, and make tea of rain water or of some kind of aerated water.

Teas and wines vary so much in their character that while some of them tend to produce constipation, others rather tend to act as laxatives. As a rule, however, tea contains a considerable proportion of tannin, which may act as an astringent; so that although in some persons tea is a laxative, yet in cases of constipation cocoa or coffee should be tried instead. Some of the red wines, such as certain clarets or ports, contain much tannin and are very astringent; white wines are less so, and in constipation are therefore to be preferred.

Exercise.—As I have said, want of exercise tends to cause constipation, and active exercise to relieve it; and that the active exercise ought to be sufficiently brisk to induce active play of the diaphragm and abdominal muscles. The languid saunter along a road, which is sometimes known by the name of a "constitutional walk," is of comparatively little service in this respect. I repeat, however, that sometimes exercise, as in cases of ovarian or uterine irritability, has just the opposite effect.

Massage.—In chronic constipation massage sometimes proves a very useful means of treatment. It may be applied to a certain extent by the patient himself, who may gently rub the abdomen in the direction of the hands of a watch every morning or night, or may roll a cannon ball (from 3 to 7 lbs. weight, and covered with chamois leather) in the same direction. Seven pounds seems a great weight when held in the hand, but a cannon ball of this weight does not feel at all too heavy when it is rolled over the abdomen. These simple methods, however, are by no means so efficacious as the regular application of massage by a trained operator. Vibration may be applied either by the hand of a masseur or by a vibrator worked by hand or by electricity. The mechanical vibrator consists of an apparatus by which a disc usually about 1½ or 2 inches in diameter is made to percuss rapidly over the large intestine.

Electricity.—The constant or the faradic current or both combined may be used. When the constant current is employed, one large electrode, say 8 by 6 inches, may be placed over the lumbar region and another of 2 or 3 inches in diameter moved over the colon from right to left. A constant current with frequent interruptions may be used for five to ten minutes at a time every day as strong as the patient can stand it. For the faradic current a coil with a rather short and thick secondary spiral is used and an interruptor with a pendulum arrangement for regulating the frequency of the interruptions. The current may either be applied

to the abdomen by two electrodes as already mentioned, or one large electrode may be placed under the back, and the other pole may be connected to an electrode in the rectum or to the arm of a masseur, so that the current passes to the abdomen through his hand during massage, and the effects of massage and electricity are thus combined (*vide* also Vol. I. p. 469). For the galvano-faradic current De Watteville's apparatus may be used in the same way as the faradic. Morton's currents of static electricity employed in a similar fashion are also useful.

Hydropathy.—In some cases hydropathic treatment is very useful. The essentials of the hydropathic treatment are the application of wet compresses to the abdomen two or three times a day and sitz baths—cold in the summer and the chill taken off in winter. These must be followed or accompanied by exercise and drinking water; so that it is almost impossible to get the full effect of the therapeutic measures in a patient who is being treated at home.

Drugs.—The drugs that have been used for the relief of chronic constipation are many indeed; but, on account of the important part which inaction of the rectum plays in the malady, aloes, which has an especial action upon that part of the bowel, is more universally employed, either alone or in combination, than any other, in the medicinal treatment of constipation. It forms a part of every purgative vegetable pill in the Pharmacopoeia with the exception of the compound scammony pill. When given alone it is convenient to administer it in the form of granules which contain one-tenth of a grain each of aloin made up with sugar of milk. One of these granules taken with each meal suffices to keep the bowels regular. One objection that is frequently raised against aloes is that it tends to cause piles; and there can be no doubt that a full dose of an aloetic purgative is very likely to bring on an attack of piles in those liable to this ailment. But small doses of aloes, on the contrary, by maintaining a regular action of the bowels, tend to prevent piles by avoiding the local pressure in the rectum which large and hard motions occasion. In combination with rhubarb or colocynth, aloes is employed as a dinner or breakfast pill, and such a formula as the following is very useful: — R Pil. colocynth. co., Pil. rhei co., āā gr. j.; Ext. hyoscyam. gr. ss. M. Ft. pil. An old friend of mine, to whom I owe this formula, told me that he had used it every night of his life for forty years; it had done him no harm: on the contrary, it had done him much good, and the long continuance of it did not seem to diminish its effect nor had the dose to be increased. He took his pill every day five minutes before dinner, so as to give it a slight start of the food, and this plan, as judged by its results, seemed to be a good one. Where the pill taken at night does not act quickly enough so as to produce a motion in the morning at the time that is usually most convenient to the patient, it may be taken at lunch or even at breakfast. The three remedies that are most in vogue at present as laxatives are the sulphur lozenges of the Pharmacopoeia, which we owe

to Sir Alfred Garrod, compound liquorice powder, and cascara sagrada. Two or three sulphur lozenges every night frequently suffice to produce a soft pultaceous action next morning; and half a teaspoonful to a teaspoonful of liquorice powder in half a wineglassful of water has a similar action in cases in which the sulphur alone is insufficient to produce the effect. Cascara sagrada, in the form of the extract or liquid extract, has largely displaced other purgatives. The solid extract is the more convenient, as it may be given in pill in doses of one to three grains either alone or combined with extract of hyoscyamus, or with extract of hyoscyamus and some hepatic stimulant, such as euonymin or iridin. A plan which is useful in some cases is to put a pinch of senna leaves or pods in a small muslin bag, and place this in a stewpan with some water and prunes. During the process of stewing the active principle of the senna is dissolved by the water, and imparts stronger aperient power to the prunes and their juice. The bag is removed when the prunes are stewed, and a little experience teaches the patient the proper quantity of the confection to use. Within the last few years various synthetic purgatives have been introduced: purgatin (anthrapurpurin acetate), exodin (a synthetic oxyquinone closely resembling purgatin), and purgen (phenolphthalein). In atony of the intestine strychnine or nux vomica is a useful adjunct; and belladonna not only tends to prevent griping, but seems to have a regulating action upon the nervous system of the intestine itself. In one case of obstinate constipation I succeeded in obtaining natural action for some months by the use of a pill containing extract of hyoscyamus and extract of nux vomica without any direct purgative whatever. The idea of using such a pill in chronic constipation was to try to shift the balance of excitability from the inhibitory to the motor nerves of the intestine. Such treatment, or treatment by small doses of opium, is, I think, especially useful in cases in which there is some reflex inhibition of the intestinal movements, and in which exercise increases constipation (*vide* p. 643). In another case of obstinate constipation in a gouty patient, I succeeded in obtaining regular action of the bowels, which had previously resisted very powerful purgatives, by putting the patient on a course of salicylate of soda. In some patients meat and beef-tea appear to have a strongly constipating action; in others they have just the reverse. In many patients the omission of meat from the diet table causes the bowels to move regularly—a result not due simply to an increased bulk in vegetable diet, for in some cases it is obtained when the diet remains otherwise almost unaltered.

In some cases, again, bread seems to have a very constipating action, and one patient, who suffered from obstinate constipation, told me that since he gave up bread entirely and lived upon fish, meat, and vegetables, his bowels have been perfectly regular.

The addition of iron appears sometimes to aid the action of aperient pills; and the pill of aloes and iron of the British Pharmacopœia sometimes acts very satisfactorily. When one aperient ceases to act another may be given: and by changing the purgative from time to time regular

action of the bowels may be maintained. Many people think that the continued action of purgatives is unnatural and injurious, but, at any rate, the harm they do is less than is likely to occur to the patient from accumulation of faecal matters in the intestine; and dinner pills may be simply looked upon in many cases as supplying the stimulants of which the food has been deprived during the process of cooking. If there be any large accumulation of hardened faeces in the bowels the faeces should not be driven down forcibly by a violent purgative, but should be washed out from below by means of an enema. Enemas consisting of warm water alone, of soap and water, or of castor oil and starch mucilage, are frequently employed, not only to remove hardened faecal masses but also to induce daily action of the bowels. It is usually said that their continued use is apt to produce a catarrhal and atonic condition of the intestine; moreover, they are troublesome and often inconvenient to the patient. Of late years enemas of one or two fluid drachms of glycerin, or suppositories containing glycerin, have been employed to cause evacuation of the bowel; and although they too are liable to lose their power, they are frequently very useful indeed, because the stimulus to defecation comes from the rectum; if no faecal matter is present no desire is felt. The advantage of glycerin is that, if a patient on going to the closet is unable to evacuate, by using glycerin he will probably get the desire, and obtain evacuation in about ten minutes, instead of waiting until next morning to obtain evacuation by means of a pill or aperient draught.

When large masses of scybala have accumulated in the intestine, it may be necessary to remove them mechanically, by breaking them down with the handle of a spoon if they are in the rectum, or by washing them out if they are higher up. In order to do this they should be softened as far as possible by the introduction of large enemas having either oil or water as a basis. Olive oil may be used alone, a pint or more being thrown up if the patient be able to retain it. Water at about a temperature of 98° F. should be used for the enema, as either cold water or very hot water tends to increase the action of the bowel. It should be introduced under very gentle pressure indeed, either with a syringe or, still better, by an irrigator, which should be suspended 12 or 18 inches above the patient's hips. The exact height must be regulated by the person's feelings; if the bowel be very irritable the pressure must be diminished; if it be but slightly irritable, the pressure may be increased. As large a quantity of water as can be retained should be thus introduced — usually from 1 to 3 pints; and it is often retained better when introduced through a soft rubber tube, not directly into the rectum, but into the sigmoid flexure. The size of the tube must be regulated according to the case; but frequently a stout tube half an inch in diameter is better than a small one, as it is not so likely to kink in the rectum, and the end of it passes more easily into the sigmoid flexure. Linseed tea, or thin starch paste, may be employed instead of plain water. In order to allow of the introduction of such large

quantities into the bowel, it is advisable to pass the rectal tube through a piece of dentist's rubber which may be held tightly against the anus, either by the fingers of the nurse or by a napkin folded round it. When desire to defecate comes on during the introduction of an enema its administration should be stopped for a few minutes, and gentle pressure on the anus kept up. The desire to defecate then usually passes away, and its cessation may be aided by very gentle rubbing over the abdomen. After the liquid has been introduced, it is well to keep a folded napkin over the anus with a certain amount of pressure for some time, so that the liquid may be retained and gradually soften the scybala. If the bowel be very irritable a dose of opium may be given—either by the mouth or, still better, as an opium injection, or as a morphia suppository—half an hour before the introduction of the softening enema.

In some cases in which faecal concretions are very hard to remove, the action of the enema below may be aided by washing out the stomach. This operation seems to exercise a stimulating action through the nerves on the movements or secretions of the intestine, and thus aids in the removal of the faecal mass.

Surgical Treatment. — The appendix vermiformis is very generally regarded as a relic of a former stage in the development of man which is now not only useless but dangerous, and its removal has become very common. Professor Metchnikoff considers that it is not merely the appendix which is useless, but that "the whole large intestine must be regarded as one of the organs possessed by man and yet harmful to his health and his life," and its presence "in the human body is the cause of a series of misfortunes." It is a reservoir of waste and putrefying material, from which poisons are formed, and in cases of constipation "certain products are absorbed by the organism and produce poisoning, often of a serious nature." He considers "that not only the rudimentary appendage and the caecum, but the whole of the large intestine are superfluous, and that their removal would be attended with happy results." This proposal has met with approval at the hands of a practical surgeon, Mr. Arbuthnot Lane, who says, "the ideal primary operation would be the removal of the large bowel to the junction of the ileum, but against such a measure there is the very unsatisfactory state of the patients."

In view, however, of the risks of such an operation, Mr. Lane contents himself with treating chronic obstinate constipation by making an anastomosis between the small intestine and sigmoid flexure or rectum, so as to dispense with the ascending, transverse, and descending colon. The short-circuited large intestine is left in situ, and is said to cause no trouble, provided the patient attends to a daily evacuation. If he does not do this, material may pass back into the caecum. This can usually be removed by an enema, but if the condition recurs, Mr. Lane removes the caecum and ascending colon up to the flexure. This further operation, he says, is a simple one, and is readily borne without risk or anxiety.

Possibly in years to come the operation recommended by Metchnikoff, and regarded as the ideal one by Mr. Lane, may be one in common use.

LAUDER BRUNTON.

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DIARRHOEA

By Sir Lauder Brunton, M.D., F.R.S., and Eustace Smith, M.D., F.R.C.P.

Bacteriology

By C. Slater, M.B.

The Bacteriology of Diarrhoea.—Numerous micro-organisms, by their direct action or indirectly by means of their toxins, are capable of producing a greater or less degree of inflammation of the gastro-intestinal tract which manifests itself as diarrhoea. The bacteriology of such forms of diarrhoea as occur in the course of clinically well-defined diseases is fairly well known, though even here it is doubtful how far the clinical disease coincides with a single bacterial cause; thus, many cases which are ranked as "typhoid" are found not to be due to the *B. typhosus* but to a group of allied though distinct organisms. The causation of the clinically ill-defined diseases which are grouped together by their most prominent symptom, diarrhoea, still, however, remains obscure, notwithstanding that they have been the subject of much investigation. This obscurity is due in part to the large number of organisms found in the diarrhoeic stools, often the only material available for investigation, and the consequent difficulty in determining which, if any, is the specific cause of the disease. The difficulty is increased because many of the organisms found are normal inhabitants, or closely resemble normal inhabitants, of the intestine whose virulence is known to be increased by the conditions found in enteritis, and there is frequently no such generalisation of any one organism in the blood or internal organs as would indicate its causal connexion with the diarrhoea; even when such generalisation does occur care must be taken not to confuse a terminal infection with a primary cause. The discovery of more delicate methods for distinguishing between micro-organisms and the consequent separation into species of bacteria at one time regarded as identical make it difficult to gauge the value of evidence obtained before these methods were employed. The specific agglutinating action of the blood-serum in cases of infection seemed likely to assist in determining the causal factor, and there is no doubt that much has already been learned, and much may be hoped for in the future from this reaction; but our knowledge is not at present sufficient to make it a safe indicator of the presence or importance of a particular microbe, as was found by the Rockefeller investigators with

regard to *B. dysenteriae*. One method of obviating some of the difficulties mentioned is to study the causation of well-marked groups of infectious diarrhoeas in order to see whether any general conclusion can be reached which can be used to explain the occurrence of less well-marked but clinically similar cases in which the proof of causation is less clear.

Diarrhoea may result from exogenous or endogenous infection, may be due to the entrance of an extraneous pathogenetic germ, or be due to such alterations in the condition of the intestinal tract or its contents as enable a normal intestinal micro-organism to produce pathogenetic effects. The infection is derived, usually, but not always, through the medium of food, from a pre-existing case of disease either in man or one of the lower animals. Occasionally food, originally quite sound, may be the seat of changes produced by saprophytic organisms (e.g. *B. botulinus*) which render it highly toxic. While it is not denied that symptoms of gastro-enteritis may be produced by "ptomaines," there is a growing opinion that such cases are rare, and that the decompositions produced by the usual organisms of putrefaction do not often cause diarrhoea (cf. art. "Food Poisoning," Vol. II. Part II. p. 855).

Though in the case of many diarrhoeas it is not at present possible to indicate the specific organisms which produce them, it is possible to shew that the normal metabolic processes of the intestine are the result of a very delicate adjustment of conditions, and that it is probable that many of the admitted causes of diarrhoea can be expressed in bacteriological terms and act by disturbing this adjustment. This seems to be especially the case with those forms of diarrhoea which by some authors are grouped as *simple fermentative or non-inflammatory*. Among the causes of the constancy of normal intestinal decompositions the preponderating influence of the normal intestinal flora and the chemical changes produced by it which render the intestinal tract unsuitable for the growth of other microbes must be reckoned. Escherich, Tissier, and others have shewn that with breast-fed infants it is justifiable to speak of a normal flora of the intestine, and even when the diet is less simple there is still considerable constancy in the organisms found in the faeces. The *B. coli* found under normal conditions appears to be especially adapted to and modified by its host, as is shewn by the specific though weak agglutinating action of the host's serum on his own bacilli (Kraus and Pfaundler) which is not exercised on similar bacilli from other intestines. In diarrhoea the number and especially the kinds of organisms in the faeces increase, the normal *B. coli* increases in virulence (Cumston), and aberrant typhoid-like races of colon bacilli appear.

The acid reaction of the normal intestinal contents is due to the presence of acetic, lactic, and succinic acids (Macfadyen, Nencki, and Sieber). Butyric acid is always abnormal (Ewald and Boas), and, as Dr. Jordan has shewn, produces shedding of the intestinal epithelium and acts as a general poison. By means of their acid products, according to some observers, but, according to Bienstock, by a direct antagonistic action of the micro-organisms, *B. coli* and *B. lactis aerogenes* prevent the

putrefactive decomposition of albumin by *B. putrificus* and probably also by other organisms, and the consequent formation of toxic bodies. It is certain that *B. putrificus* taken by the mouth disappears from the intestine, and that while this organism will cause the putrefaction of sterile milk, ordinary milk containing *B. coli* is not affected by it ; a point not without interest in treatment.

In diarrhoea there is increased decomposition of albumin, as indicated by increased phenol, indol, sulphuretted hydrogen, and methyl-mercaptan in the faeces, and it has been shewn that the faecal bacteria in diarrhoea produce more active fermentation than those from normal stools, while abnormal products such as butyric acid appear. Acting on this knowledge Escherich has shewn that in the follicular enteritis of children, which is accompanied by much putrefaction in the large intestine and the production of alkaline stools, the administration of carbohydrates acts beneficially, while on the other hand affections of the small intestine with abnormal increase of acids are favourably influenced by a strictly albuminous diet. Considerations of this kind, too, have led Metchnikoff to recommend a lactic acid diet, and induced Tissier to suggest the use of cultures of *B. acidi paralactici* and *B. bifidus* for intestinal disorders.

The *B. coli* of the normal intestine is not, as held by Lesage and Macaigne, a harmless organism, and its non-pathogenicity is attributed in chief part to the protective action of the uninjured and living intestinal epithelium which prevents the absorption of the toxin and the invasion of the tissues by the bacillus. Any lesion may permit this absorption of toxin with the consequent production through vasomotor influences of hyperaemia, haemorrhage, and desquamation of the epithelium with consequent further facilities for absorption. It is obvious then that without the introduction of any specific pathogenetic germ, the normal balance may be upset by the ingestion of food containing large numbers of bacilli, as in the case of unclean milk in summer, by the use of food containing easily fermented bodies or foods which have already undergone fermentation, and contain either toxic chemical bodies, such as butyric acid, or fermentative products in such quantities as to alter the usual reaction of the gastro-intestinal tract or produce actual changes in its wall. Changes in the rate of passage through the gut from increased peristalsis, in the amount of intestinal secretion, in the condition of the intestinal wall produced through vasomotor or nervous mechanism, will all tend to the production of diarrhoea through microbic agency.

The specific diarrhoeas produced by such organisms as *B. choleraе*, *B. typhosus*, *B. tuberculosis*, need not be discussed here except in so far as the well-ascertained facts of these diseases throw light on the obscurer forms. There is no doubt that these organisms may be present in the intestinal canal in a virulent form without producing the corresponding diseases. The importance of the condition of the intestinal wall and contents is well marked in the case of cholera (Pfeiffer), and the increase of the colon bacilli of the intestine as the result of a typhoid infection illustrates the influence of one microbe on the activity of another.

The organisms which are most commonly associated with the diarrhoeic diseases of both adults and children belong to the colon group, or are organisms very closely allied to members of that group, and have characters intermediate between those of *B. coli* and *B. typhosus*.

The *B. coli communis*, on experimental grounds and on account of the frequency with which it is found, often in a condition of exalted virulence and often in almost pure culture, was at one time, and especially by French observers, regarded as of extreme importance as a cause of diarrhoea. Lesage stated that 25 per cent of cases of diarrhoea in breast-fed children were due to this organism alone. It has been considered the cause of epidemic enteritis in children, and to it has been attributed the pathogenetic action of various foods, milk, ice creams, and other articles (Klein, Andrewes). The gradual recognition that there are many varieties of the colon bacillus, that there is frequently a generalisation of the microbe either shortly before or after death, and that the bacillus is rarely agglutinated except in low dilution by the serum of the patient, has led to the present opinion that the classical *B. coli communis* (Escherich) is probably not concerned with the production of diarrhoea, but that varieties, generally less active in their action on sugars, are more important. Escherich considers, however, that there is a definite disease of young children of from 2 to 5 years old, epidemic in character, occurring in autumn, accompanied by dysenteric stools, occasionally spreading to the attendants, and named by him colitis contagiosa or enteritis dysenteriformis, which is caused by the *B. coli communis*. The serum of the patients agglutinated the bacillus, which was isolated in pure culture during the height of the disease, and was very virulent at first, but soon lost its virulence. The agglutinating colonies could, however, only be found at first, were in limited numbers, and those which agglutinated in highest dilution were atypical non-fermenting varieties. Similar cases were reported by Finkelstein in Berlin, and by Valagussa in Italy, and by some American observers. Valagussa found, however, that the bacillus isolated agglutinated with a dysentery serum in 1 : 50 dilution, and that the same serum acted therapeutically, and the cases were considered to be true dysentery. One of the forms of the dysentery bacillus was also found in many of the American cases. The *B. coli communis* is still, however, thought to be responsible for many outbreaks of diarrhoea by Charlton and Jehle and others who are fully alive to the danger of mistaking this bacillus for an allied organism.

The *B. enteritidis* (Gärtner). — A large number of meat-poisoning epidemics marked by acute gastro-intestinal symptoms, though at one time considered to be caused by ptomaines, have been definitely traced to infection by *B. enteritidis*, or closely allied organisms. Recognised by Bollinger in cases of "sepsis intestinalis," the specific organism was isolated by Gärtner in 1888, and since then numerous epidemics have been reported in England and abroad in which this organism was found. In at least four-fifths of the epidemics the cause has been the ingestion of the flesh of diseased animals, especially of such as have suffered

from septic inflammatory processes, puerperal or traumatic septicaemias (metritis, mastitis), or symptoms of enteritis. The micro-organism has been isolated from the incriminated food and from the faeces and internal organs of the patients. Agglutination in high dilution occurs with the serum of the patient, and this plays a large part in the diagnosis and epidemiology of the outbreaks. The organism is extremely pathogenetic, and produces highly toxic products which resist heating even to 100° C. Though very closely allied the organisms isolated from different epidemics shew small variations, and, as differentiated by their agglutinating properties, have been arranged by de Nobele into two groups—(1) the *B. enteritidis* group, and (2) the *B. aertrycke* group, to which the organisms isolated by Dr. Durham belong. An investigation of the carcases of animals which have died from the diseases mentioned above as being especially liable to render the meat dangerous, has not led to any constant or frequent discovery of organisms allied to *B. enteritidis*. Basenau has found occasionally in meat the *B. bovis morbificans* which appears to belong to this group, as does also an organism found in a special septicaemia of calves (Thomassen). Probably the disease of the animal is important chiefly because it produces conditions which favour the escape of organisms from the intestinal canal into the tissues, and possibly also increase the virulence of the invading microbe. Though in the majority of these well-marked epidemics the flesh of diseased animals has been the cause, it is important to note that the meat, originally healthy, may become infected, or may become contaminated by faecal or other matter containing the *B. enteritidis*, as was shewn by Prof. Delépine in the Derby epidemic of 1902. Water, and through this oysters, according to Dr. Durham, may be similarly contaminated, as also may milk, probably from the evacuations of cows with enteritis. Dr. Klein has found *B. enteritidis* in a considerable number of samples of milk. There also appear to be cases of direct infection from man to man (van Ermengem). The interest of these epidemics in connexion with diarrhoea lies in the fact that these cases of acute enteritis can be definitely referred to a single organism or group of organisms, while clinically there is nothing to differentiate them from the acute epidemic enteritis of children or from sporadic cases. It seems reasonable to suppose that these latter forms of diarrhoea are also infections, and since experience shews that they are often connected with food, that the infection may be of the same kind as that in meat poisoning. This is rendered the more probable by Mr. Morgan's important observation that bacilli of the enteritidis group (Aertrycke type), and others allied to the paratyphoid bacilli, are to be found in the normal intestines of food-yielding animals, and have been found in the intestine of a child dead from bronchopneumonia. Karlinski has also found them four times in normal faeces. These bacilli of meat-poisoning epidemics are then very widely spread intestinal bacilli, scanty in normal intestines, more numerous in inflammatory conditions, and apparently also more virulent. It is found that the search for these bacilli—and the same observation

applies to the *B. dysenteriae*—is more successful when scrapings of the intestinal mucous membrane instead of faeces are examined. This seems to imply an intimate relation between the bacilli and the mucous membrane. Infected food which has been kept for some time after cooking is especially dangerous, and this would seem to shew that the number of bacilli is important in these infections, or that these bacilli require the help of preformed toxin, as in the case of *B. tetani*, before they become dangerous to the healthy intestine. Possibly the less massive bacterial infection of food may explain the difference between the food-poisoning epidemics and the less fulminant outbreaks of diarrhoea.

B. paratyphosus and B. paracoli.—These meat-poisoning epidemics have also been linked to enteric fever by the discovery that the paracolon and paratyphoid bacilli originally isolated from cases of supposed typhoid fever belong to the "intermediate group," and have been found in cases of food poisoning by Schottmüller, Trautmann, Levy and Fornet, and others. Trautmann has also connected an epidemic septicaemia of rats with the *B. paratyphosus* β, and Klimenko has found it in the faeces of healthy dogs. These observations have still further increased the significance of the organisms of the "intermediate group" in the etiology of diarrhoeic diseases.

B. dysenteriae; Bacillary Dysentery.—The organism isolated by Shiga in 1898 from cases of acute dysentery in Japan is now practically accepted as the cause of this disease, and also, if the organism described by Flexner and Strong be accepted as a variety of Shiga's bacillus, as the cause of epidemic and sporadic cases in temperate regions.

Asylum or Institutional Dysentery.—In 1900 Drs. Mott and Durham shewed that it was not possible, on clinical or pathological grounds, to separate the disease known as "asylum dysentery" from the acute form; and in the same year Kruse isolated from asylum cases a bacillus identical with that found by Shiga. Since then Drs. Eyre and M'Weeney in Great Britain, and observers in America, have also demonstrated the presence of Shiga's bacillus in the asylum disease.

Acute Epidemic Enteritis.—In 1902 Duval and Bassett of Baltimore found the Flexner-Strong variety of *B. dysenteriae* in cases of summer diarrhoea of infants, and a combined investigation undertaken by the Rockefeller Institute confirmed and extended their conclusions. In over 60 per cent of the cases of summer diarrhoea of children the bacillus was found. The strain of organism was usually the Flexner-Harris, but the Shiga strain, as well as several other variants, were also found. In many cases the bacillus isolated is agglutinated by the patient's serum, but Flexner is careful to point out that the occurrence of agglutination is not to be treated as an index of the presence of, or infection with, *B. dysenteriae*. Though in many cases the *B. dysenteriae* was alone isolated, not unfrequently other organisms, and especially streptococci, were associated with it. It was impossible to associate definite clinical varieties with special races of dysentery bacilli, and more than one variety might be present in the same case (Knox and Schorer). The opinion was

formed that the Shiga strain was found in the most acute cases, and that mixed infections were more fatal than simple. Besides the two strains of dysentery bacilli mentioned, there have been separated from clinically similar cases other varieties of bacilli which are characterised as a rule by greater fermentative action on sugars, and are regarded by some bacteriologists as variants of the type bacillus, and by others constituting a group of paradysentery bacilli (Park). One organism found in summer diarrhoea, the *Bacillus B.* of Duval and Schorer, approaches the colon bacillus in character. If these organisms be all regarded as dysentery bacilli, then dysentery bacilli are found in a very large number of cases of diarrhoea, at any rate in America. At the same time the specific character of the disease produced is to a large extent lost, and the changes in the intestine are reduced to a minimum. Bacilli of the Flexner type have been isolated from the stools of healthy children to whom cathartics had been administered and even without this treatment (Duval and Schorer, and Charlton and Jehle). European observers have not as yet confirmed the results obtained in America. Charlton and Jehle describe cases of summer diarrhoea which they attribute to the *B. coli communis*, and cases of meat poisoning in which *B. dysenteriae* was found. In twenty-eight out of fifty-eight cases of summer diarrhoea of children Mr. Morgan has found a bacillus which is not identical with any of the dysentery bacilli described, and he further found that he was unable to obtain, with forty-four samples of blood tested, any agglutination of bacilli of the accepted strains. These results are in strong contrast with those of American observers. The dysentery bacilli appear to form a group of closely allied organisms, which, like those of the enteritidis group, are probably normally present in the intestine, and in intimate relation with the intestinal mucosa. That they are capable of producing acute dysentery seems to be fairly well proved, but they are found also in cases of less severity when their relation to the disease is less definite.

The bacteriology of acute epidemic enteritis is far from being definitely settled. There is but little doubt that it is of bacterial origin, as every factor which increases the probability of bacterial infection, whether directly or through food, is known to increase the probability of the occurrence of this disease. It is impossible to do more than mention the influence of temperature, of breast-feeding as opposed to bottle-feeding, of good milk as against milk bacteriologically bad, of flies as contaminators of food, or to do more than simply refer to the work of the New York Board of Health, of Professor Delépine at Manchester, and of many others on the connexion between the disease and the bacterial contamination of food.

Looking at the facts mentioned above as a whole, it seems that a very large number of diarrhoeas, including food poisonings, dysenteries, acute infantile diarrhoeas, and other less well-defined varieties, are infections caused by a group of allied bacilli found in the normal intestine of men or animals; and that more will be gained by the discovery of the factors which render these bacilli specially virulent, whether this be due to

changes in the micro-organism or in the host, than in determining the presence of a particular variant of a group of organisms which, like the streptococci, according to Drs. Andrewes and Horder, are not yet thoroughly differentiated into specific groups.

B. enteritidis sporogenes (Klein) has been associated with three outbreaks of diarrhoea at St. Bartholomew's Hospital, and from the number of the bacilli found in the flakes of mucus in the diarrhoeic evacuations, and more particularly because one epidemic could be traced to food infected with this organism and but little likely to be contaminated with non-sporing organisms, the *B. enteritidis sporogenes* was thought to be the cause of the outbreaks. It is a very widely distributed intestinal bacillus, and if the cause of diarrhoea, as seems probable, this organism like those bacilli which have just been described, owes its pathogenetic action to associated conditions.

Diarrhoea is not uncommon as a symptom of general infection by pyogenetic organisms, but a primary enteritis caused by streptococci and staphylococci is less frequent.

Streptococci are found in the normal intestine of the adult, but are apparently only slightly pathogenetic (Andrewes). They increase in numbers and in pathogenicity in enteritis caused by other germs, as was shewn to be the case in dysentery by Kruse and Pasquale, and by the Rockefeller investigators. In the adult, cases of primary enteritis due to streptococci have been reported by Tavel, and from cases of cholera nostras the same organism has been isolated, possessing sufficient virulence to infect a second case. Cases of fatal enteritis have been recorded by Dr. Andrewes and by Drs. Washbourn and Pakes, in which streptococci were abundantly found. In children there seems to be no doubt that a primary streptococcic enteritis, which is followed by a general blood infection, may occur. With this Booker, Escherich, and his school agree, and Canon describes similar cases. The streptococcus produces either by direct infection of the mucous membranes, or by its toxin, a severe inflammation with desquamation of the epithelium of the intestine. The disease is chiefly in the large intestine. The stools are numerous, and at the height of the disease have a bloody muco-purulent character. Streptococci have also been found in cases of infantile summer diarrhoea, and four epidemics were attributed by Holst to the *Streptococcus longus* found in the milk of cows suffering from mastitis.

The *Staphylococcus pyogenes aureus* seems to be of much less importance, but is considered by Moro to be the cause of the intestinal catarrh of breast-fed infants.

B. pyocyaneus was found by Thiercelin and Baginsky in the intestinal canal of sucklings associated with fever and diarrhoea, and Lartigau found it in adults in an epidemic resembling dysentery. Calmette's work on the endemic enterocolitis of Cochin-China appears to prove that this organism can, under certain conditions, cause dysentery. In epidemic enteritis in children it has been found in a virulent form by Knox and Schorer. Ehlers has found it in the heart-blood of a child

dead of enteritis, and Escherich held it to be the cause of gastro-enteritis in sucklings.

Capsulated bacilli, such as *B. friedlaenderi*, and many organisms of the Proteus group have also been associated with diarrhoea.

Peptonising bacilli have been regarded as the cause of gastritis in nurslings (Flügge), and Lubenau states that *B. peptonificans* was the cause of an epidemic of gastro-enteritis, while Robertson considers that peptonising organisms are the chief cause of summer diarrhoea.

Diarrhoeas caused by Protozoa.—Various diarrhoeas, especially those occurring in the tropics, are associated with, and in some cases possibly caused by, the presence of protozoa in the bowel or stools. The organisms found are: (1) Amoebae: *Entamoeba coli* and *Entamoeba histolytica* (Schaudinn); (2) Flagellata: *Trichomonas hominis* (Davaine), *Lamblia intestinalis* and *Entamoeba undulans* (Castellani); (3) Ciliata: *Balantidium coli* and *B. minutum*; (4) Coccidia: *Coccidium cuniculi* and *Coccidium hominis*. The diseases associated with these protozoa are described in the article on Dysentery (*vide* Vol. II. Part II. p. 544 *et seq.*).

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Diarrhoea.—The name "diarrhoea" is usually employed, and properly so, to indicate a fluid and too frequent discharge from the bowels. Although there may be three or four evacuations in the day, yet the term "diarrhoea" is not usually applied to them if they be properly formed and not liquid. Sometimes, however, when patients complain of "diarrhoea" we find, upon inquiry, that the motions are formed, but that there is a desire to go too frequently to the closet. Constipation is the commonest ailment of civilised countries, but diarrhoea destroys more lives than any other disease. The name "diarrhoea," like that of "constipation" or of "dyspepsia," simply denotes a symptom which may depend upon many different causes.

Causes of Diarrhoea.—Diarrhoea is due to an unusually watery condition of the intestinal contents at the time when they reach the rectum and are evacuated. This watery condition may depend upon:—

(A) Diminished absorption of water by the intestine; (B) increased secretion of fluid from the intestinal mucous membrane; or (C) a combination of both.

Diminished absorption is the commoner cause; and it may be due to the intestine having (*a*) less absorbing power, or (*b*) less time in which to absorb. The contents of the small intestine when they reach the caecum are normally quite fluid; but during their sojourn in the large intestine the watery parts become absorbed to a greater or less extent; so that generally they become firm, and before they reach the rectum are moulded into cylindrical masses. Sometimes, indeed, absorption goes further, and they become not only firm, but dry and hard. It is obvious that conditions which affect the small intestine alone will hardly cause diarrhoea. An irritable or catarrhal condition of the small intestine may exist, and its contents may be hurried on with great rapidity into the

large intestine, yet, if the peristaltic movements of the caecum and colon be not increased, the whole of the fluid parts of the faeces may be absorbed, and the motions may be natural or even constipated.

On the other hand, any irritation of the large intestine which results in increased rapidity of its peristaltic movements will tend to produce diarrhoea, by hurrying on the liquid faeces from the caecum to the anus so quickly that too little time is afforded for the absorption of the watery constituents. If both the small and the large intestine have their peristaltic movements increased, diarrhoea will of course be greater than if either were affected alone.

Forms of Diarrhoea.—*Diarrhoea from Purgatives.*—A temporary diarrhoea may be induced by the administration of purgatives. Many of these—like colocynth, senna, cascara, castor oil, and croton oil—affect the muscular coats of the intestine, and especially of the colon; while they have little or no effect upon the processes of absorption or secretion. They simply quicken the peristaltic movements and hurry the fluids which are poured into the caecum by the ileum so quickly on through the large intestine that there is not time for their watery constituents to become absorbed.

Other medicines, again, especially neutral salts, greatly increase the secretion of fluid from the intestinal mucous membrane. This is shewn by the fact that if three loops of intestine be isolated by means of ligatures, and a solution of a neutral salt be injected into the middle loop, and the whole be returned to the abdomen of the animal, secretion takes place to a large extent into the middle loop; so that, if the animal be killed some hours afterwards, the quantity of fluid contained in this loop will be found to be greatly in excess of the saline solution injected into it, while the loops on each side of it remain empty. This experiment shews conclusively that a large secretion of fluid has taken place from the intestinal mucous membrane under the stimulation of the saline solution. Some salines, however, while producing a large secretion of fluid, hardly stimulate peristaltic action at all, and thus have no purgative effect. For example, we know that acid tartrate of potash injected into a loop of intestine will cause secretion; and in a patient who has taken a dose of this medicine secretion probably takes place abundantly in the small intestine, as shewn by the gurgling and rumbling in the bowels; yet the salt stimulates the peristaltic movements of the large intestine so slightly that the whole of the fluid which has been rumbling about may be absorbed, and the subsequent motion, instead of being loose, may be somewhat constipated. Accordingly, when we wish to sweep out the intestinal contents thoroughly, we combine drugs which will act upon the mucous membrane with those which will stimulate peristaltic movements; we employ, for example, the compound jalap powder, in which the acid tartrate of potash is intended to produce watery secretion, the jalap to stimulate the peristaltic movements, and the ginger to act as a carminative and prevent griping.

Diarrhoea from Food.—An action upon the intestinal secretion and

movements similar to those which are caused voluntarily by means of purgative medicines are frequently produced involuntarily by various substances taken as food, or by the products of their digestion or decomposition in the stomach and intestines. Thus, a meal too abundant in quantity, instead of being vomited, may pass through the pyloric orifice, and by irritating the intestines give rise to several watery discharges: after this, the intestinal canal, being freed from its incubus, may return to its normal condition. At other times the products of an imperfect digestion may act as purgatives. Thus, fatty acids formed in the stomach, instead of producing vomiting, may cause purging; and too long a delay in the stomach will probably render laxative even such substances as milk; for we find that milk which has been artificially over-peptonised, and thereby rendered bitter, frequently causes looseness of the bowels in patients who take it. Other articles of food may contain definite poisons; for example, certain specimens of cheese contain a toxamine, tyrotoxicon (Vaughan) [see art. "Food Poisoning," Vol. II. Part I. p. 865]. Poisonous mushrooms also contain a poison, muscarine, which causes vomiting and purging; and even esculent mushrooms contain a substance called amanitine, which, although harmless in itself, occasionally undergoes change and yields the poisonous muscarine; thus now and again violent vomiting and purging may result from the use of mushrooms which are apparently good.

Diarrhoea from Microbic Poisons.—Within the last few years it has been shewn that albuminous substances, during the process of decomposition by microbes of various kinds, yield many poisons, amongst which are muscarine and other allied substances having a similar action; so that protein substances, which have been imperfectly digested and have undergone putrefaction in the intestine, may be converted into powerful poisons, and give rise to violent diarrhoea.

Faeces contain an immense number of micro-organisms; Sucksdorff has estimated the number of bacteria passed daily in the stools of one person as amounting on an average to 53,124,000,000. No wonder, then, that when faeces are retained too long in the intestine they may act as irritants, and that scybalous masses may frequently give rise to diarrhoea, which ceases on their removal. [The bacteriology of diarrhoea is considered on p. 653.] The diarrhoea of tropical countries is in all probability dependent upon micro-organisms, although the exact nature of these has not been as yet determined (*vide* "Sprue" and "Hill Diarrhoea," Vol. II. Part II. pp. 545, 567).

Diarrhoea from Entozoa.—Entozoa present in the intestine, such as tapeworms, round-worms, or threadworms, may sometimes give rise to diarrhoea. This may be due partly to the mechanical irritation; but in the case of the tapeworm it seems to be partly due also to irritant substances secreted by the parasite.

Uraemic Diarrhoea.—Various poisons which circulate in the blood may be eliminated by the mucous membrane of the intestine in the same way as already described in the case of the stomach; and as in the latter organ they may give rise to vomiting, so in the intestine they may

occasion diarrhoea. It is probably to the elimination of toxalbumins through the intestinal mucous membrane that the persistent diarrhoea in cases of kidney failure and threatened uraemia is due.

Diarrhoea from Chills.—There can be no doubt whatever that exposure to cold, and especially to cold affecting the abdominal walls, will give rise to diarrhoea; but the mode of this action is not yet precisely ascertained. Probably the chill gives rise to an acute catarrh in the intestinal mucous membrane of the patient, just as it would cause catarrh of the respiratory passages in others; but we cannot say with certainty how far the diarrhoea is due to changes in the nervous system, in the circulation, or to tissue-metamorphosis giving rise to purgative products.

Nervous Diarrhoea.—Mental emotion has a powerful effect upon the intestines, so that fright frequently causes defecation, and if continued may give rise to diarrhoea. There is a certain form of nervous diarrhoea which is often very troublesome: so long as the patient is within easy reach of a closet, and can empty his bowels whenever he feels the desire, he is perfectly well; but the moment he becomes aware that there may be difficulty or delay in evacuating his bowels, he feels the desire to do so.

Malarial Diarrhoea.—This is probably to be reckoned as a form of nervous diarrhoea. In some patients it recurs regularly at the same hour of the day, resists all ordinary astringents, but yields readily to the administration of quinine.

Morning Diarrhoea.—An obstinate form of diarrhoea is sometimes met with in connexion with irritability of the sigmoid flexure, and sometimes also with dilatation of the stomach; it is very often known as "morning diarrhoea." The patient has to get up very early in the morning, and has perhaps three to six movements before 11 A.M. After this he is pretty free, and probably after mid-day he has no action at all until next morning. This form of diarrhoea is sometimes not so severe, and occurs only in the shape of one movement, just after breakfast, followed by one or two loose motions within the next hour or so, but both the severe form and the mild are apt to resist medical treatment. Morning diarrhoea is also common in persons addicted to alcohol.

Diarrhoea due to a Prolapse of the Sigmoid Flexure into the Rectum.—This diarrhoea is chronic and irritating, the motions are small, but they are numerous and often accompanied by tenesmus. The rectum in the adult is pretty firmly fixed to the bones of the pelvis, especially to the sacrum; so that, although prolapse of the rectum through the anus is common in children, it is by no means common in adults. But prolapse of the sigmoid flexure into the rectum is by no means uncommon in adults; sometimes it gives rise to constipation, but frequently it causes chronic diarrhoea. My attention was very strongly drawn to this disorder by a case that I saw some years ago in a tea-planter who had come from Assam. He had suffered from chronic diarrhoea for from six months to a year before I saw him. The whole of his trouble dated from an attack of violent constipation which had lasted many days. To relieve

this he took a strong purgative, and from the moment that this acted he had suffered from diarrhoea. All the remedies he had tried had failed. On examination by the bowel I found a state of things which simulated a prolapsed rectum in a child; but in this case the prolapse was as far up the rectum as the finger could reach. High up in the rectum I found pendent bags of mucous membrane, and in the middle of these bags I could feel the opening into the sigmoid flexure.

Malignant Disease.—To one other cause of diarrhoea I must draw attention, and that is malignant disease of the bowel (*vide* p. 751). For diarrhoea due to lardaceous disease of the intestines see p. 569.

Symptoms.—The symptoms of diarrhoea depend very much on the part of the bowel affected, and the extent and nature of the affection. I have already said that irritation or catarrh of the small intestine does not necessarily lead to diarrhoea; but that if it be associated with an irritable condition of the large intestine, so that the contents of the duodenum are rapidly swept down through the whole intestinal tube, the faeces are not unlikely to contain a certain amount of altered bile which either gives a green colour to the faecal masses, or betrays itself in a yellow or greenish tinge of the mucus which accompanies the motion.

In obstruction of the common bile-duct the motions may be perfectly colourless. In such a case the urine and conjunctivae are usually deeply tinged with bile; but if the pancreatic duct alone be obstructed the motions may still be perfectly colourless, and yet no jaundice be present, nor the urine be tinged with bile. In such cases, notwithstanding their pale colour, the faeces actually contain bile; but the colour is obscured by undigested fat which usually comes from the milk or cream in the diet. In sprue the motions are generally frothy. In some cases, from decomposition of the intestinal contents, the faeces have a most abominable smell. The exact nature of the substances to which these smells are due has not been determined; but similar substances are readily produced by digesting fibrin with active pancreas at the temperature of the body for about eighteen hours.

When the diarrhoea is severe, the discharges excessive, and the motions abundant and watery, the blood becomes drained of its watery parts, circulation becomes feeble, there is a tendency to venous stagnation, and signs of general collapse may appear. These are most marked in cholera (Vol. II. Part II. p. 435), but may be noticed also to a slighter extent in ordinary diarrhoea, and especially in children. The pulse is small and feeble, the skin pale and cold, the face shrunken, the eyes sunken in their orbits; cramps may attack the legs; and in children, on account of the lowered blood-pressure, the fontanelle is greatly depressed. On account of the frequent passage of the motions, great tenderness, soreness, and excoriation of the anus, and even of the nates, may occur. The irritation which gives rise to diarrhoea is frequently accompanied by pain, and this may be so excessive as to lead to faintness or actual syncope, especially in persons who suffer from a feeble heart. Even when pain is not present diarrhoea is apt to produce or be accompanied

by malaise, languor, irritability sometimes, and, if excessive, by general prostration.

Chronic diarrhoea dependent upon local irritation in the rectum may go on for a long time without any indication whatever of general depression, emaciation, or disturbance of any kind, beyond the simple annoyance which is caused to the patient by repeated calls to the closet. In cases in which much mucus is passed, and especially, I think, in those cases in which the sigmoid is prolapsed into the rectum, a condition of hypochondriasis is very frequently observed. In any case there is great depression of spirits and inability to take any interest in external things; the attention of the patient is concentrated upon his own condition, and more especially upon the state of his bowels. In many cases of this sort mucus is passed in large quantities, either surrounding the motion or separately; and sometimes the amount is so great that it appears to form a regular cast of the bowel, and has been described as membranous enteritis. Two varieties of mucous diarrhoea have been described as membranous enteritis and mucous colic. In the first of these there are recurrent spasmodic pains; in the second there is more generally a feeling of continued discomfort and uneasiness, frequently accompanied by a tendency to nervous depression and hypochondriasis (*vide* p. 816). The causation of these conditions has not been precisely ascertained; and I am inclined to believe that in many cases they depend upon prolapse of the sigmoid into the rectum.

In cases such as I have described there is generally a sense of incomplete evacuation of the bowels, even soon after the motion has passed; and the more the patient strains the worse this feeling is apt to become, because the prolapsed part of the bowel becomes more congested.

Ulceration of the rectum gives rise to diarrhoea, and also sometimes to a desire to sit long at the closet. I am inclined to think that such ulceration occasionally occurs from mechanical injury to the mucous membrane of the bowel by the bone or ivory nozzles of enema syringes; to avoid this it is better to use a soft india-rubber tube, which is not likely to hurt the bowel.

In some cases there is a constant desire to defecate, although there be nothing whatever present in the bowel, and nothing can be felt wrong with its wall. I have seen one case in which the desire was so incessant that the patient, an old woman, sat permanently on a bedpan; the only opinion that I could form was that the eczema, from which she suffered, had affected the mucous membrane of the intestine, and gave rise to a constant irritation in the rectum similar to that which it caused in the skin around the anus.

Usually diarrhoea is either accompanied or preceded by intestinal pain; but this is not invariably the case. The pain sometimes occurs in the small intestine, but more usually in the colon; hence its name of colic. Like those other pains—such as hepatic and renal colic—to which the name of colic has been extended, which also occur in organs containing involuntary muscular fibre, the pain in the intestine probably depends upon irritation to the intestinal nerves, partly by cramp-like contractions of the

involuntary muscular fibre in some sections of the bowel, partly by excessive dilatation in other sections. Such pain may occur without diarrhoea, because the cramp-like contractions tend rather to interfere with the passage of the intestinal contents than to accelerate them; in lead colic the pain is very marked, but is usually accompanied by obstinate constipation. In migraine the pain in the head is associated either with excessive contraction or excessive dilatation of the arteries, and, sometimes at least, with dilatation of the proximal and contraction of the distal ends of the arteries supplying that part of the head in which the pain is felt. In this ailment there seems to be at the same time a dilatation of the proximal, and a contraction of the distal, end of the stomach, the fundus being much dilated and the pylorus firmly contracted. It is probable that a like condition occurs to some extent in the intestine, and either in consequence of this, or of contraction of the intestinal vessels, similar to what occurs in the branches of the carotid in migraine, a pain comes on in the intestine similar to that of headache. In some persons the two come on together; but in others the pain appears sometimes in the head and sometimes in the stomach, the stomach being free when the head is painful, and conversely. In certain cases diarrhoea occurs along with headache, and appears to replace the vomiting which usually accompanies migraine.

Treatment.—The treatment of diarrhoea consists, first, in removing any irritant which may be giving rise to it; and, secondly, in soothing any irritation of the intestine itself which may remain after the irritant has been removed. In the case of artificial diarrhoea, due to purgatives, great care must be taken to ensure their removal; and thus it is the common practice to give a saline purgative next morning after a dose of calomel. This practice is not only useful in ensuring the removal of waste products from the intestine, but of clearing out the calomel itself; sometimes if the calomel be taken alone at night, and no saline in the morning, the calomel will act as a powerful irritant, giving rise to much pain: in one case which I saw it nearly produced collapse, very much as if a dose of corrosive sublimate had been taken; and similar results sometimes arise from a colocynth pill or from proprietary pills.

In the case of diarrhoea caused by food, either excessive in quantity, objectionable in quality, or so decomposed in the intestine as to be irritating, one of the best means of stopping the diarrhoea is to give a dose of castor oil together with a little opium—as, for example, half an ounce of castor oil and 8 minims of tincture of opium. The castor oil clears out the intestine thoroughly, irritant matters are removed, and the opium soothes the bowel and allows it to recover from the irritation. If, in place of a purgative an astringent be given, the movements of the bowels are temporarily arrested, but the irritant remains there; and, if it consist of decomposing food, its retention makes matters worse, so that after a day or two the diarrhoea returns in a severer form than before.

Another favourite remedy for diarrhoea due to indiscretions in diet is compound rhubarb powder; the rhubarb and magnesia act as purga-

tives and clear out the intestine, while the slight amount of tannic acid contained in the rhubarb acts subsequently as an astringent.

When the diarrhoea is not due to irritating substances in the bowel, but to inflammation of the mucous membrane of the intestine itself, as in the catarrh which succeeds violent irritation, one of the most common remedies is chalk mixture. Lime forms with fatty acids an insoluble soap, and will thus neutralise such acids, which are strong intestinal irritants. It will also neutralise other acids not of the fatty series, and by its combination, either with acids or albumin, will tend to form a non-irritating pellicle over the mucous membrane; while at the same time lime salts slow the movement of involuntary muscular fibre, and tend to lessen peristalsis. At the same time the carminatives present in the chalk mixture aid the expulsion of flatus, and prevent the pain which would otherwise arise from its distending the bowel. When the pain is severe, and chalk mixture alone is insufficient, its combination with opium in the form of the pulvis cretae aromaticus cum opio of the Pharmacopoeia is exceedingly useful; or 5 to 10 minims of tinctura opii may be added to the ordinary chalk mixture. It is hard to explain the effect of brandy in checking simple diarrhoea; but there can be no doubt that it is the most useful remedy, and an ounce of neat brandy is a most useful remedy in simple diarrhoea. Its efficacy may be increased by the addition of 5 to 10 minims of tincture of opium, or 20 minims of chlorodyne: these remedies indeed often prove very useful in checking diarrhoea without brandy, or anything else. It must be borne in mind, however, that if the diarrhoea depend upon irritating substances, the use of these remedies, while giving relief for a time, may render the diarrhoea more troublesome if they are taken before the irritant substances have been passed out. If, however, the diarrhoea have continued for some days and the irritant be already removed, the brandy and opium may soothe the intestine and effect a permanent cure.

In many cases diarrhoea is due to mixed causes, and there are both irritating substances in the intestine and an irritated or inflamed condition of the mucous membrane itself. A useful plan of treatment is, therefore, to give, first, a dose of half an ounce of castor oil with 5 to 10 minims of laudanum. An hour afterwards let the patient take some warm tea or other warm drink to assist the oil. After it has acted freely administer a sedative such as the following:—℞ Bismuthi carbonatis, sodii bicarbonatis, āā gr. x.; spt. chloroformi, ℥ x.; aq. menthae piperitae, vel aq. cinnamomi, vel aq. caryophylli, ad. ℥j. Ft. Hst. This may be given every four hours twenty minutes before food. At the same time an ounce of mistura cretae, to which 5 to 8 minims of laudanum may be added, should be given after every loose motion. This direction regulates the amount of astringent which the patient takes according to his requirements; for if the bowels are only opened once loosely he takes but one dose, whereas if there are ten loose motions he gets ten doses. For patients who are travelling, or who cannot stay at home on account of business engagements, draughts are awkward; for them

bismuth lozenges, soda-mint tablets, or 20-grain powders of pulvis cretae aromaticus, or pulv. cret. aromat. c. opio, tablets of these remedies, or 20 drops of chlorodyne are most convenient. In place of giving opium by the mouth the drug may frequently be administered with great advantage by the bowel; either as the ordinary enema opii containing half a drachm of laudanum in 2 ounces of starch mucilage, in the form of a morphine suppository, or of a compound lead suppository, in which the astringent action of the lead tends to supplement the soothing action of the morphine.

The pain which frequently accompanies diarrhoea may be greatly lessened by the application of a large warm fomentation or poultice to the abdomen. In cases in which these remedies cannot readily be applied, as on a railway journey, relief may be obtained by applying a tin or india-rubber bag containing hot water to the abdomen over the clothes. In the case of a poultice it is best not to apply it, as is sometimes done, directly to the skin, or with only a thin bit of muslin between it and the skin, for in this fashion the poultice must either be allowed to become half cold or the patient's skin will be scalded. Much more relief is afforded by putting two folds of flannel between the skin and the poultice, which may then be made boiling hot, and the heat, coming gradually through the flannel, does not burn the skin.

Carminatives frequently lessen the pain greatly by rendering the contraction of the bowel more even, lessening spasm, and assisting the escape of flatulence. A useful mixture is half a drachm of aromatic spirit of ammonia, half a drachm of compound tincture of cardamoms, and 10 or 15 minims of spirit of chloroform. Ten minims of compound spirit of ether may be substituted for the spirit of chloroform in the mixture or added to it; and if the pain be very great, half a drachm to a drachm of the compound tincture of camphor makes a useful addition. The whole of this may be given in an ounce of water, or, still better, perhaps, in peppermint water. In cases in which distension of the colon with flatus is very great, it may sometimes be relieved by passing a long flexible india-rubber tube into the colon; but usually the wind may be brought away by injecting about 4 ounces of dill-water, pepper and water, or some other carminative. Or an enema of turpentine may be employed; but probably the best of all injections for the relief of tympanitic distension is the enema asafetidae, although its disagreeable odour prevents it from being used so frequently as, from its great power of relieving flatulence, it would otherwise be.

Diarrhoea due to decomposed food, to mushrooms, or to cheese, is best treated by clearing out the bowels by castor oil; but along with this belladonna or atropine should be given, because atropine is an antidote to muscarine, the poisonous alkaloid of mushrooms, and to a substance which is likewise formed by the decomposition of albumin. Ten minims of the tincture of belladonna, or $\frac{1}{100}$th of a grain of atropine, may be given every half hour, or every hour, either until the diarrhoea is less, or till the symptoms of physiological action have begun to shew themselves in

dryness of the mouth or dilatation of the pupil. Of course this remedy must be used under constant supervision, as it is hardly safe to leave it to the patient's friends to decide when the drug is to be stopped.

In diarrhoea due to microbic infection one may combine the administration of various antiseptics, to destroy the microbes themselves, with measures for the removal of the poisons which they form. Among the most powerful of these is mercury in various forms—grey powder, calomel, corrosive sublimate, and biniodide.

The first two may be given in single doses of five grains of grey powder, or two to three of calomel; or in divided doses, such as one-third to two grains of grey powder, and one-third to one-half of a grain of calomel, every two to four hours. One thirty-second to one-sixteenth of a grain of biniodide or of corrosive sublimate, or half a drachm to a drachm of the liquor hydrargyri perchloridi, may be used in a similar way.

Bismuth, in the form of carbonate, subnitrate, or salicylate, is a useful intestinal sedative; the carbonate is the form which acts best with chalk, but the salicylate is the most powerful disinfectant salt of the three, and may be given with salicylate of soda or with salol. Salol passes through the stomach unchanged, but becomes converted in the intestine into salicylic acid and phenol, both of which are powerful disinfectants. It may be given in doses of 10 or 15 grains in a cachet, alone or with salicylate of bismuth. β-naphthol and its compound with salicylic acid, called naphthalol or betol, are both used (in doses of 5 to 10 grains) in the same way, and for the same purposes, as salol. Naphthalin is another useful intestinal disinfectant, and is one of the most powerful of them all in deodorising offensive faecal motions. In the diarrhoea of children, for example, it completely removes the offensive odour, but, so far as my own experience goes, it will not by itself arrest the diarrhoea; it has been stated by others, however, to do so.

Besides killing the pathogenetic bacteria in the intestine by means of intestinal disinfectants, they may be destroyed by the simple plan of starving them out. Although Allan MacFadyen and I found that bacteria appeared to have the power of adapting themselves to changed foods, and of manufacturing new ferments wherewith to digest them, a certain time is required for this adaptation; and if the food be rapidly and frequently changed the bacteria may be starved. Thus, when milk has been used as a diet, and the intestine has become infected with bacteria which live readily upon milk, the complete stoppage of the milk for a while, and substitution for it of farinaceous food only, will tend to kill off a large number of the bacteria which thrive on milk. After a day or two other bacteria may grow and multiply upon farinaceous food; but if this again be suddenly changed for a meat-juice, a great number of these will be killed off, and then by reverting to milk, and so on, the various bacteria may be gradually eliminated (*vide* also articles on "Cholera," "Diarrhoea of Children," "Dysentery," "Enteric Fever," "Sprue," "Tuberculosis," "Pulmonary Tuberculosis").

In the cases of ordinary diarrhoea, which are so common in persons

who have been living in India, Afghanistan, or other tropical climates, medicines are frequently useless; and the only method of cure is that proposed by Sir Joseph Fayrer, of putting the patient on an absolute milk diet with no other food whatever; the patient, in fact, is treated exactly as if he had enteric fever. In some cases the patient may be able to attend to his work and even put on flesh, but this is not usual; and, as a rule, while on the milk diet he will have to keep in bed.

Astringents of various kinds are frequently useful in diarrhoea after the acute stage has passed off, and it has become chronic. The chief astringents are some of the heavy metals in various forms. Rhubarb, as already mentioned, is useful, as after clearing away irritants from the intestine its rheo-tannic acid has an astringent action. Catechu in various forms, rhatany, logwood, are all occasionally useful. The more soluble preparations, such as infusion and tincture of catechu, expend part of their energy on the stomach before passing into the intestine; and therefore the compound catechu powder, in which the three astringents, catechu, kino, and rhatany, pass into the intestine along with two aromatics, cinnamon and nutmeg, is perhaps more useful, in doses of 20 or 30 grains. Compound kino powder in 10-grain doses is also very useful, the kino acting as an astringent, and the opium lessening pain and irritation. Various compounds of tannin are also useful: tannalbin and honthin (tannin with albumin), tanocol (tannin with gelatin), both in doses of 15 grains; tannigen (insoluble in the stomach and soluble only in the intestine) and tannoform (tannin with formic aldehyde), both in doses of 3 to 8 grains. In a very chronic case of diarrhoea the lead and opium pill is useful, and so are the sulphates of zinc and copper in doses of 1 to 2 grains in the form of pill, either alone or with opium.

In all cases of chronic diarrhoea the urine should be carefully examined, and if it be found to have a persistently low specific gravity, and to contain a trace of albumin, great care should be taken not to check the diarrhoea hastily by means of opium or astringents; in such cases the bowels form the chief channel for elimination of the products of tissue waste, and if the diarrhoea be checked the patient may quickly die of uraemia.

In cases of tendency to diarrhoea from cold, and, indeed, in all cases of chronic diarrhoea or tendency to diarrhoea, the abdomen should be kept at a warm and equable temperature, either by a flannel bandage or by a silk scarf wound several times round it. Attention should also be paid to the feet, the shins, and the back of the neck, as a chill in those spots is not unlikely to recall the diarrhoea or abdominal pain, even although the abdomen itself be kept warm.

Cases of nervous diarrhoea are to be treated by moral remedies; the patient should be encouraged to resist the inclination to defecate, just as a patient with a nervous irritability of the bladder is told to resist the desire to micturate. In the form of nervous diarrhoea in which the introduction of food into the stomach gives immediate desire to go to

stool, bismuth before meals tends to check the impulse; the same result is sometimes obtained, even more easily, by the use of liquor arsenicalis, in doses of half a minim to a minim in an ounce of water or peppermint water, ten or fifteen minutes before meals. In diarrhoea depending upon locomotor ataxy small doses of antipyrin may prove useful.

Malarial diarrhoea, although it resists the usual astringents, often yields readily to quinine or cinchona. In such cases, if the liver is much enlarged and tender, the cure may be quickened by the administration of a mercurial followed by a saline purgative; thus the congestion of the liver is reduced before quinine or astringents come into the field.

Morning diarrhoea may generally be greatly relieved and sometimes entirely checked by the simple plan of telling the patient to take no liquid after five o'clock in the afternoon; all the liquids should be taken in the early part of the day. In persons, on the contrary, who suffer from evening diarrhoea, the liquids should be taken late at night, and very little during the early part of the day. In all cases the use of alcohol should be carefully regulated.

In cases in which diarrhoea depends upon prolapse of the sigmoid flexure into the rectum, it should be treated by avoiding all articles of food which would be likely to pass through the intestine undigested and to irritate the tender part of the bowel. The bowels may be kept open by means of an enema of half a pint of water every morning; and immediately after the action an astringent injection of 1 or 2 drachms of catechu in 2 ounces of water should be thrown into the bowel and retained there. In place of the injection, ointment may be introduced by means of one of Allingham's ointment-carriers. If there be irritation in the sigmoid flexure itself, I have had an ointment-carrier, similar to Allingham's, made of a larger size; if a soft india-rubber tube be attached to it the ointment can be passed well up into the sigmoid.

In cases of very chronic diarrhoea, more especially where dysenteric or other ulcers are supposed to be present, large clysters of disinfectant and astringent solutions are sometimes useful. For this purpose a pint or more of the solution should be employed in the manner already directed for the softening of scybala. These clysters may consist either simply of warm water or preferably of normal saline (about a drachm of common salt to the pint of water) or of thin starch paste, to which are added various sedative and astringent substances, such as carbonate of bismuth (5 to 10 grains per ounce), boric acid (1 grain per ounce), salicylic acid (1½ grains per ounce), thymol (⅛ to ¼ of a grain per ounce), nitrate of silver, sulphate of copper and chloride of zinc (½ grain to 3 grains per ounce). The clyster should be retained as long as it can be with comfort. On account of the risk arising from absorption, powerful poisons like corrosive sublimate, iodide of mercury, or carbolic acid are inadmissible.

In some cases of very obstinate diarrhoea, especially of a dysenteric character, surgical aid may be of great service. The caecum is fixed to the abdominal wall, and a small opening being made into it, the bowel may be flushed out with a quart or more of warm saline by means of a

catheter introduced into the opening. The astringent solution may then be allowed to pass along the bowel in the same way. If there is haemorrhage from the bowel a solution of calcium chloride, of 5 grains or more to the ounce, may be passed through.

In cases of malignant disease of the bowel relief may be afforded by a sedative suppository such as the following:—R Ext. belladon. gr. ½, morphinae gr. ⅓, cocainae gr. ½-1 gr., ol. theobrom. *q.s.* Ft. suppositorium, or by the injection of 30 to 60 minims of tinct. opii made up to 2 fluid drachms with water and injected into the rectum by a glycerin-syringe. If the diseased part of the bowel cannot be removed by surgical interference, the bowel should be kept open by the use of a warm water enema thrown up the bowel high enough to pass beyond the obstruction and soften the faeces above; so as to prevent any hardened mass coming down upon the constricted portion and thus giving rise to sudden obstruction. When obstruction threatens an artificial anus must be made; but when diarrhoea is due to malignant disease of the descending colon or of the sigmoid, relief may be afforded by opening the abdomen and connecting the bowel above and below the diseased portion, so that the faecal matters pass by the affected part instead of through it. The operation of complete excision of the diseased part with union of the ends of the intestine has not been very successful hitherto; but if surgery improves as much within the next quarter of a century as it has done in the last, it is possible that such an operation may be frequently and successfully performed, and patients cured in cases and under conditions at present regarded as hopeless.

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L. B.

The Diarrhoeas of Children.—In the young child—especially in the infant with his feeble hold upon life—a looseness of the bowels may be a matter of no little moment. It is, indeed, to this derangement that a large number of the deaths which occur in infancy are due. The growing child is more dependent than the adult upon a continuous supply of food; for, with a poorer blood, he has to carry on a more considerable work. The adult has only to maintain the current nutrition of the body; the child, while doing this, has also to provide material for growth and development. His organs, therefore, are always kept hard at work; and that they may work to good purpose, the blood must be maintained at a normal standard by a regular inflow of nutritive material. When the supply is suddenly interrupted, as must happen in any derangement which gravely affects the digestive functions, the blood at once becomes poor and nutrition flags. If to this state of oligaemia be added the exhaustion induced by a copious drain from the bowels, the consequences to a young infant may be disastrous.

Diarrhoea is a symptom, and as such may be met with in every degree of severity from a simple looseness of the evacuations, which causes little reduction of strength and yields readily to treatment, to a profuse watery drain which exhausts the tissues of fluid and leads to death in a few hours. The age of the patient, the state of his general health, the cleanliness of his surroundings, the cause or causes to which his illness is due, and the pathological conditions which accompany it, are all matters which may have a very definite influence upon the course and issue of the complaint.

Etiology.—In its simplest form diarrhoea is the consequence of a mild catarrh due to direct irritation of the intestinal mucous membrane, or to exposure to cold. In hand-fed babies looseness of the bowels is often excited by unwise feeding; for the food, as it passes undigested along the alimentary canal, ferments and sets up catarrh of the mucous membrane. This is especially apt to occur in nurseries where the supply of food for the whole day is prepared in the morning, as fermentative

processes soon begin in the sweetened mixture. But it is not infants only who suffer in this way, for in children of all ages irritability of bowel may be caused by improper food. The looseness is, no doubt, an effort of the digestive canal to rid itself of the offending matter; and if the dietary be amended at once, the stools soon recover their normal appearance.

Chilling of the surface is another cause of simple diarrhoea. Infants when first short-coated often suffer in this way. Sometimes it is in the nursery bath that the chill is contracted, for with many nurses it is a matter of principle to reduce the temperature of the bath water to the lowest point the child can bear without shewing signs of distress; and in a susceptible child a prolonged bath in water which is merely tepid will often set up the complaint. The rapid alternations of temperature so often met with during an English summer—the temperature falling suddenly 10° or 15°—are almost always followed by an outbreak of diarrhoea amongst the children. Rickety subjects, probably on account of their profuse perspirations, are especially prone to suffer from these atmospheric changes; and in teething infants the process of dentition, as it excites a certain amount of fever, is often accompanied by a mild relaxation of the bowels. In the latter case it is to the pyrexia, and not to the accidental cause of the pyrexia, that the complaint must be ascribed. The child suffers not because he is cutting a tooth but because he is feverish, and therefore keenly susceptible to changes of temperature. But even a mild purging is usually something more than a mere catarrh of the bowel. The relaxation is accompanied by fermentative processes which are due, no doubt, to the action of bacteria.

The same causes which set up a simple diarrhoea in a healthy child will often induce a much graver disturbance in one whose nutrition is faulty, or who is living under imperfect sanitary conditions. But the worst forms of diarrhoea occur during the summer heats, and seem to be the direct consequence of the heightened temperature aided, no doubt, by septic changes in the contents of the bowel. It has long been held that the prevalence and fatality of the summer diarrhoeas have a definite relation to the degree of heat; and Dr. Dawson Williams has brought together much valuable evidence tending to shew that the prime element in the etiology of the illness is a high minimum temperature. Dr. Henry Tomkins was the first to urge the value of exact observations upon the temperature of the earth, for heat which has sunk for a few feet into the ground is parted with but slowly, and a heated soil must do much to maintain the temperature of the air. He found that as soon as the thermometer registered 59° to 62° F. at a depth of one foot below the surface the causes producing the disease began to operate. Dr. Ballard pushed the inquiry to a depth of four feet, as at this distance from the surface the temperature rises and falls more slowly still. He states that in summer the mortality from bowel complaints does not begin until at four feet the thermometer records a temperature of 56° F. This statement has been confirmed by Dr. J. Priestley, who shewed that the rise in

the diarrhoea death-rate followed a rise in the earth temperature to 56°, and fell again as the heat of the soil sank below that point.

In its worst forms, such as occur mainly in children under two years of age, summer diarrhoea is rarely seen out of large towns, and is most virulent in seasons when great heat is combined with drought. It is at such times that, owing to the deficient rainfall, the sewers and drains are imperfectly flushed and often foul; and decomposition in the retained organic matter is promoted by the high temperature. Heat seems also to have an influence in rendering the bowel secretions favourable to the multiplication of noxious organisms and increasing their capacity for mischief. The bacteriology of diarrhoea is treated of at length elsewhere (p. 653) and need not here be repeated. It may, however, be stated that in the case of young infants the more virulent microbes seem to be derived from tainted milk and foods long prepared and stale. In a dangerous form the complaint is rarely seen in breast-fed children who live under good sanitary conditions and draw their whole nourishment from the breast, sucking the milk straight from the gland without risk of contamination. If the disease be ever seen under such conditions, it is probable that other food or drink is being given to the child, either from heedlessness or in order to make up for deficiencies in the breast-milk; and it must be remembered that even water from a neglected cistern, unless freshly sterilised and given from a perfectly clean vessel, may be a possible conveyor of germs to the patient. To put a stop to this summer scourge to infant life it is of the first necessity to insure the purity of the milk-supply. Cow's milk may be delivered at the house in an unsound state, and even if fresh and unpolluted will soon undergo fermentation if received into dirty jugs. The large milk companies are now taking elaborate precautions to prevent contamination of the milk while it remains under their control, and the smaller dealers are at last becoming impressed with the necessity for care in this matter. The indifference to cleanliness commonly shewn by the ordinary farm labourer, and the filthy state in which the cows often return from their pastures and stand about in their stalls, is a matter of common observation, and has, no doubt, always been the chief source of impurities in the milk.

Inflammatory conditions in the cow may also be a source of contamination. Holst in four separate epidemics of acute gastro-intestinal catarrh found the *Streptococcus longus* in milk supplied by cows who were suffering from mastitis. He states that these cocci, when injected into mice and rabbits, set up diarrhoea, and when swallowed by himself caused colic and vomiting. In serious cases the ill effects produced by the microbes are not mere fermentative processes as in the milder varieties of diarrhoea; they seem rather to depend upon decomposition of the protein constituents of the milk and the formation of noxious chemical compounds; these when absorbed give rise to the toxic symptoms. Certainly in the more virulent cases the whole course of the illness points to an infective origin, especially as the patient often dies, not merely exhausted by the drain from the bowels, but in a state of nervous collapse, which may continue

for many days after the character and number of the stools has ceased to be a cause for anxiety. There is every reason to believe that the more virulent forms of infective diarrhoea are capable of being spread by contagion. It has been asserted that noxious organisms have been detected in the air surrounding the cots of infants who are suffering from it. If this be the case, milk exposed in the wards might conceivably become infected. Probably, however, the complaint is more often spread through carelessness on the part of nurses who, after changing soiled napkins, have not properly disinfected their hands before distributing food to the patients.

The septic variety of diarrhoea is most common during the first two years of life, but it is seen also with fair frequency at a later period of childhood. It attacks strong subjects as well as weakly ones: indeed, a condition of sturdy health, although it may influence the issue of the illness (but even this is doubtful), seems to afford little protection, if any, against its onset. This cannot be said of other non-septic forms of purging, however serious they may be. As a rule weakness in the child —a condition which implies a lowered power of resisting hurtful influences —tends to favour the occurrence of inflammatory diarrhoea; and in many diseases a looseness of the bowels is a familiar complication. In enteric fever, measles, and scarlatina, diarrhoea is common: all forms of catarrhal complaints may be accompanied by it; and causes which set up congestion of the portal system, such as cirrhosis of the liver and diseases of the heart and lungs, lead to obstruction in the whole venous system and help to induce it.

Morbid Anatomy.—In the child the lesions are much less constant than is the case with older persons, and it is often a matter of surprise that after death from a severe attack of bowel complaint the morbid appearances should be so ill-defined. This curious want of correspondence between the serious symptoms observed during life and the slender traces noted later in the bowel may be seen both in acute and chronic cases. Inflammatory redness and swelling, patches of false membrane, haemorrhagic extravasations, and more or less deep ulceration of the mucous membrane may all be present; but we never feel certain that we shall find them. We see them in one case; in another which presents symptoms apparently identical we look for them in vain. Ulcerations of the bowel are more common in the subacute and chronic cases than in those which run a shorter course, and are often suspected when not actually present. In the worst forms of choleraic diarrhoea the most marked changes are seen not in the bowel but in distant parts of the body. Anaemia of the brain and other organs is common; but sometimes there is hyperaemia of the meninges and cerebral cortex due, possibly, to the action of microbes or of the poison they originate. In the chronic form of diarrhoea meningeal haemorrhage may occur, or the dural sinuses may be blocked by thrombosis from infective phlebitis. Marfan attributes all these lesions to microbic agency which sets up tox-infective processes, and states that chronic affections of the meninges and encephalon, such as chronic hydrocephalus and cerebral sclerosis, may arise from an attack of acute gastro-enteritis

in infancy. Of other organs the kidneys are apt to be the seat of parenchymatous nephritis. Often the liver is fatty, and in cases of entero-colitis, when the illness is of some weeks' duration, this fatty change is seldom absent. In addition, patches of bronchopneumonia may be found in the lungs. Sevestre has drawn attention to this complication, and attributes it to direct infection. In four out of five cases of bronchopneumonia complicating enteritis, under the care of this physician, Lesage obtained pure cultures of the *B. coli* from the patches in the lung.

ACUTE DIARRHOEA.—**Varieties.**—For convenience of description three forms of acute diarrhoea in infants and children will be described: A, simple non-inflammatory diarrhoea (mild intestinal catarrh); B, inflammatory diarrhoea (acute entero-colitis); and C, choleraic or septic diarrhoea (infantile cholera). Chronic diarrhoea will be discussed afterwards (p. 697).

A. **Simple Non-Inflammatory Diarrhoea.**—This variety is a mere disorder of function due to a mild intestinal catarrh. The purging yields readily to treatment if the conditions are favourable; but if it be aggravated by insanitary influences it may pass into one of the other varieties, and may then prove fatal.

Symptoms.—In infants the looseness may begin quite suddenly, or be preceded for some hours by signs of indigestion and abdominal pain. The tongue is furred and the child may retch or even vomit. He is peevish and restless, and as he cries draws up his legs uneasily as if griped. At this time the temperature may be elevated a couple of degrees or so, but there is usually a fall after the first discharge from the bowels. The earlier motions consist of thin faecal matter containing small lumps of undigested curd. They are usually sour-smelling or even frothy from fermentation. As the discharges continue they become thinner and, if the lower bowel be the part chiefly affected, they often contain visible mucus. They are then passed with some straining, and a streak of blood may perhaps be noticed on the diaper. The motions are often coloured green. This, according to Pfeiffer, is due to the conversion of the bilirubin of the bile into biliverdin, owing to an abnormal alkalinity of the contents of the bowel. In this case the stools contain a large quantity of bile, and their reaction is neutral or only faintly acid. But according to some observers there is a distinct variety of green stool which is contagious. It owes its colour, they say, to a special pigment which is generated by a rounded bacillus. Lesage even states that he has succeeded in cultivating this microbe and in setting up a green diarrhoea in animals by injecting it into the blood. This experience has not as yet been confirmed by the experiments of others; but I think many clinical observers may be disposed to agree with Lesage as to the occasional contagiousness of an apparently harmless diarrhoea.

At first the motions are passed frequently and in large quantity; but soon they become less numerous and only number five or six in the twenty-four hours. As a rule griping is not noticed after the first few

dejections, and there is no tenderness or swelling of the belly. The child, however, evidently suffers in his nutrition; his colour fades, and his flesh very quickly becomes soft and flabby. This form of looseness appears to be due to direct worry of the mucous membrane, and tends to subside of itself when the irritating matter has been discharged. If in warm weather it persist beyond a very few days, it may pass into a more serious form.

In older children the symptoms are much the same as in infants: but as the patient is stronger his nutrition suffers less, and although he may look pale he loses little flesh. His tongue is furred, but unless the stomach is disordered as well as the bowels the child is in good spirits and takes his food well: indeed, if not troubled with abdominal pain he will not allow that he is ill.

There is a special form of looseness of the bowels to which children are subject at the age of five or six years and upwards. It is called "lienteric diarrhoea," and appears to be due to an exaggerated peristaltic action of the bowels, for the stools follow directly upon a meal with great urgency and are preceded by severe abdominal pain. The child may be noticed to turn pale soon after sitting down to table, and often rises and hurries from the room before the meal is at an end. The same thing occurs whenever food is taken, and, in addition, the bowels may act in the early morning, or when the child first leaves his bed. The motions consist of undigested food and mucus. Sometimes the pains are complained of without being followed by a stool; and sometimes, although the bowels act with urgency after a meal, abdominal discomfort seems to be absent. The tongue is slightly furred, or is red and irritable-looking with projecting papillae. This form of looseness interferes greatly with nutrition. The act of eating seems to rouse the muscular coat of the bowel to excessive movement: it is not that the stools are numerous but that they contain, little changed, nearly all the food that has been taken. The child may eat heartily enough, but the meal is carried so quickly along the alimentary canal that the process of digestion can hardly begin. As a consequence the patient soon becomes pale and languid and thin; and if the derangement continue, as it often does for weeks together, his evident loss of flesh causes the greatest anxiety amongst his friends.

B. **Inflammatory Diarrhoea (Entero-Colitis).**—Inflammatory diarrhoea is a much more serious complaint than that just described. While it lasts, not only is nutrition put a stop to, but the patient's little stock of strength is reduced by high fever and a profuse watery diarrhoea. The disorder, therefore, is a serious trial even to a sturdy child, and often proves fatal to feeble children and infants. In such patients symptoms of depression come on early if the purging be severe, and, moreover, in infancy there is a special tendency to deficient action of the kidneys which adds greatly to the danger of the illness.

This form of diarrhoea is associated with evidences of inflammation, and sometimes, in protracted cases, of ulceration of the intestinal mucous

membrane. It may begin suddenly or gradually, and often follows upon an attack of mild intestinal catarrh, or is a sequel of the septic form of diarrhoea to be afterwards described. When established its chief features are a high bodily temperature with frequent and watery discharges from the bowels. It is difficult to say whether all the symptoms are due to the anatomical changes noted in fatal cases, or whether unwholesome fermentations in the bowels and the absorption into the system of poisonous ptomaines may not be answerable for some of them. I believe that this noxious decomposition is present at any rate in the cases where early signs of collapse are noted, or where the course of the illness is exceptionally rapid and severe.

Symptoms.—As a rule the first symptoms are those of an intestinal catarrh. In infants there is some abdominal pain, as shewn by cries and uneasy movements of the legs; and the loose stools consist of undigested and fermenting matter. If there be any gastric catarrh the child vomits also. After the food contained in the bowels has been evacuated the stools get thinner and more offensive, and are tinted brown or green. Often they number ten, fifteen, or twenty in the day and night, and the more frequently they are passed the thinner they become. Usually they vary in character from time to time, and, although always very offensive, are thin and pasty, or frothy and dark, or consist of a greenish watery fluid which deposits a small quantity of thin faecal matter. So long as the lower bowel is unaffected there is no mucus visible to the naked eye. Under the microscope they are found to consist of particles of casein, starch granules, fragments of meat-fibre, epithelial and round cells, many varieties of bacteria, and enormous quantities of fat.

The purging may be accompanied by fever from the first, but often at the beginning the temperature is normal, and the attack has all the characters of an ordinary intestinal catarrh. Sooner or latter, however, the temperature rises to 102° or 103° F. or higher, and the general symptoms at once become severe. The child wastes quickly and grows very weak. His face is pale; his eyes get hollow; and the corners of his lips are encircled by a deep wrinkle like the mouth of a very old man. The tongue may be a little furred, but is usually clean and red and inclined to be dry. Sometimes vomiting is distressing; the pulse is then very rapid and feeble and the exhaustion great. The skin generally is dry and may be reddened about the buttocks and backs of the thighs by the irritation of the discharges from the bowels. The excretion of urine is scanty and, if the stools are profuse and watery or the vomiting urgent, no urine may be passed for twenty-four hours at a stretch, or even for several days. When this is the case I have known general oedema to occur. We must not, however, necessarily infer, because no urine is passed, that the kidneys have ceased to act, for sometimes the bladder is found to be distended with urine which it cannot discharge, either from want of contractile power or a spasmodic state of the sphincter. Extreme distension of the bladder has been shewn experimentally in the dog to be capable of causing general oedema by raising

the pressure in the ureters and kidneys, and so diminishing excretion. (Oddo and Sarles.)

In a few days the depression of the child becomes extreme, and his fontanelle sinks deeply. His pulse is quick and very feeble; his breathing is hurried and shallow; his eyes are hollow; the lids are purple and close incompletely, and his face is livid. There is usually great thirst, and the tongue is dry and brown, or may be sprinkled with thrush. The feet and hands often feel cold although a thermometer placed in the rectum registers 105°, 106° F., or even higher. I have known the mercury to rise as high as 109° in an infant who afterwards recovered; but such an extreme temperature is a very grave symptom, and if the heat mount rapidly from a moderate to a high level the patient often dies in a state of collapse.

When the inflammatory process affects especially the larger bowel (colitis) each stool is preceded by griping pains in the belly, and accompanied by much effort and straining. Often the bowel prolapses: and when the straining is urgent the protrusion may be difficult to replace. The stools appear gelatinous from excess of mucus, and may be streaked or strained with blood. On this account the complaint is often spoken of as "dysentery" or "dysenteric diarrhoea," and not without reason, for it is stated that these cases are especially prone to be associated with the *Bacillus dysenteriae* of Shiga, and that the organism is found in them in large numbers. As a rule in this country such symptoms are easily controlled, but a bad case is met with from time to time in which the vomiting is distressing, the abdominal pain frequent and severe, and the tenesmus almost constant. The prolapsed mucous membrane then bulges from the body like a bright red glistening ball, and is shot out again at once when an effort is made to replace it. The stools are small and frequent, and consist of little else than mucus, either clear or tinged more or less deeply with blood. In this form the illness approaches the type commonly met with in tropical climates. The temperature may be high and the depression extreme, and the attack often ends fatally. If with high fever the vomiting is obstinate and the tenesmus urgent, we have reason to fear the worst.

In fatal cases of inflammatory diarrhoea death is almost always preceded by the signs of general collapse above described. In certain cases this condition may be due to the occurrence of parenchymatous nephritis; but it is exceptional to find obvious signs of inflammatory mischief in the kidneys after death. I believe, however, that an arrested excretion of urine and consequent imperfect depuration of the blood does often occur without leaving recognisable traces of organic change; for whenever there is a marked tendency to drowsiness and collapse the skin begins early to lose its elasticity. This change is best noticed on the abdomen where the skin on being pinched up lies in loose wrinkles as left by the finger and thumb. The urine in such cases is very scanty and difficult to obtain, but Kjellberg states that it is sometimes albuminous, and deposits epithelium and hyaline casts and small round cells. The child is very drowsy

and stupid, but, if severely griped, may shew signs of abdominal pain. Usually, however, he seems to suffer little if at all, but lingers on—perhaps for days—in a dull, apathetic state, careless of all that passes around him, until he ceases to breathe. In very young babies the drowsiness may deepen into coma, and the infant fall into a state which has been called "spurious hydrocephalus," from its likeness to the third stage of meningitis. Lethargy, at first partial only, soon becomes profound; the child lies straight on his back, with pinched, sharp features and a leaden-coloured face, breathing irregularly and sometimes heaving a sigh. His eyes seen between the half-closed lids are dull-looking; and the pupils are sluggish and often unequal. The fontanelle is deeply sunken, and if the infant be very young the bones of the skull can be felt to overlap. The nose, the feet, and the hands are cold, and sometimes the legs feel chilled as high as the hips. In the rectum the temperature is normal or below the level of health. In this state small greenish watery stools may still be passed without effort, but often when the coma begins the purging ceases. The most energetic stimulation will usually fail to rouse a child so profoundly depressed, and life is rarely prolonged beyond a very few days. Sometimes death is preceded by a convulsion. These symptoms are usually ascribed to a sluggish state of the circulation in the brain, but they are probably in many cases the effect of profound septic intoxication.

After the age of infancy the complaint, although still a dangerous one, is yet of less gravity than before. The child has greater strength to face the weakening effects of the illness and runs less risk of grave complications. The nutrition of the patient, however, is at once arrested. He begins straightway to lose flesh and strength, his face becomes haggard, and his eyes are hollow. If vomiting be present also the features get pinched, the expression more distressed, and wasting goes on faster than before. There is usually thirst, and a craving for fluid may lead the patient to swallow beef-tea, milk, and other nourishing liquids; but he has no appetite for food. The child is often uneasy and restless, but there seems to be little actual pain unless the lower bowel be affected. The stools are watery and dark in colour, or may be yellow or green from excess of bile. Often they contain lumps of curd and masses of fat, and have a very offensive odour. The urine is high-coloured and scanty, and may contain excess of indican. If vomiting be urgent the renal excretion may be almost suppressed. In such cases the skin becomes markedly inelastic, especially over the belly; and the abdominal wall itself is often so sunken and hollow as to resemble that of a case of tuberculous meningitis. The temperature is either elevated throughout the attack, as in the case of younger children, or falls after the first few days to a level below that of health. If the case end badly death is generally due to exhaustion; therefore a patient who is already weak when the attack begins has necessarily a less chance of recovery. For this reason inflammatory diarrhoea, occurring as a complication in the course of another illness, must always give rise to much anxiety.

The *duration* of acute entero-colitis is usually short. A fatal attack rarely lasts longer than a week, and often comes to an end on the third or fourth day. The immediate danger is, of course, in proportion to the violence of the symptoms; but it does not follow that a case which begins mildly must always end in recovery. The illness may drag on for days or even weeks, varying in intensity, but gradually bringing the child lower and lower until he sinks from exhaustion. These, it must be owned, are usually cases in which the patient is regularly washed or exposed in other ways to chill. On the other hand, we sometimes see the attack begin violently enough with high fever and frequent watery stools: but after a day or two the temperature falls, the purging abates, and all danger is at an end. These abortive attacks are sometimes described as a special variety of the complaint. Of patients who survive some mend quickly; others are left pale, feeble, and subject to catarrhs; others, again, suffer for months, and even die at last of a chronic intestinal derangement which is often complicated with intestinal ulceration, and sometimes dependent upon it.

Complications.—There are certain intercurrent lesions which may be met with, especially in the more protracted cases, and add to the danger of the illness. Simple and parasitic stomatitis are not rare, and increase the difficulty of feeding the patient. Pulmonary catarrh often comes on; and as the child becomes exhausted, collapse of the lung is apt to occur, and helps to shorten his life. Catarrhal pneumonia has already been referred to. According to Lesage it is due to direct infection of the lungs by breathing air tainted with the virulent *Bacillus coli*. As it greatly increases the weakness of the patient and hastens the end the pulmonary inflammation at the time of death is usually still in an early stage. On this account it often passes unheeded, especially as the great weakness of the patient forbids complete examination of the chest.

A few twitching movements may be noticed before the end, but are not common. Sometimes grave nervous symptoms occur early in the illness. I have seen a fair number of cases—generally in children of two or three years of age—in which the patient, after several days of vomiting and purging, not necessarily of a very urgent character, has been taken more or less suddenly with convulsions, has then sunk into a state of coma and died without any recovery of consciousness. Some of these cases may have been due to septic meningitis, but where an examination of the body could be obtained it was uncommon to find evidence of intracranial inflammation. They were probably the consequence of intense septic poisoning.

An acute otitis media sometimes arises in the course of an intestinal affection, and may set up serious cerebral symptoms. Therefore in all cases in which such symptoms appear the ears should be examined with a speculum and reflected light. I have known delirium, convulsions, rigidity of the muscles of the neck, and a condition approaching coma to cease quickly when a perforation of the tympanic membrane has allowed pent-up pus to escape from the ear cavity.

Diagnosis.—Before pronouncing a case to be one of inflammatory diarrhoea we must be careful to exclude other feverish complaints which sometimes start with digestive troubles. Pneumonia and some of the eruptive fevers may begin in this way. The skin then must be searched for signs of rash, and as we proceed with our examination the early symptoms of measles, scarlet fever, and the like must be kept in mind. An acute entero-colitis is not often confused with enteric fever during the first few years of life, for the latter complaint is rare in early childhood, and when it occurs is accompanied more often by confined bowels than by diarrhoea. In the older children, however, there may be some uncertainty upon the point; but in entero-colitis the abrupt onset and shorter course of the illness, the severity of the purging from the first, the pinched expression of the patient, and, as time goes on, the absence of rash or of splenic enlargement, should be sufficient to exclude the specific fever. It may be remarked that a "pea-soup" look of the motions for a day or two is in itself no proof that the complaint is typhoid. Stools of this kind are not uncommon in the diarrhoeas of children, and we must not be deceived by them, especially as a trained nurse may cause some trouble by confidently pronouncing the case on this ground alone to be one of enteric fever.

The seat of the inflammation in the bowels may often be inferred from the appearance of the stools. Nothnagel has shewn that mucus is always present in the evacuations, and, when invisible to the eye, can be detected by the microscope. The higher in the intestinal tract the inflammation is seated the more intimate is the admixture of the mucus with the faecal contents of the bowel. In a catarrh limited to the upper part of the bowel no mucus is visible to the naked eye, but it can be detected by the microscope. In such a case the colon may be taken to be healthy. If under the microscope the mucus is seen to be tinted with bile we infer that the jejunum and ileum are also implicated; the presence of bile indicates also that there is increased peristaltic action of the muscular coat forcing the contents along, for if the bile linger in the bowels it becomes changed in appearance and ceases to respond to Gmelin's test. If mucus be plainly visible to the naked eye the lower bowel is always affected. Pulpy stools containing dabs and drops of free mucus point to the colon; and pure mucus in large quantity to the sigmoid flexure. Scybala imbedded in mucus shew catarrh of the rectum, in which case painful tenesmus is always present.

It is scarcely possible in a case of entero-colitis to be quite sure as to the exact pathological condition. Ulceration is probably present in the more protracted cases only, and even in these must not be too hastily assumed. Blood-stained stools, as Dr. Donkin has well pointed out, cannot be held by themselves to imply actual breach of surface, since oozing of blood from a merely congested mucous membrane is far from uncommon. Still, attacks of colicky pain preceding the stool, with some increase of tension of the abdominal wall, are symptoms which should excite inquiry. If, in addition, there be moderate tenderness on deep pressure along the

course of the colon, and especially if small dark clots can be detected in the stools, ulceration of the larger bowel may be more than suspected.

Prognosis.—An attack of inflammatory diarrhoea is always a serious thing, and if the complaint occur in an infant, especially in one whose digestive organs are continually overtaxed and teased by ill-chosen food, disastrous consequences may be expected. The prospect, therefore, is less bright in infancy than in later life, and is gloomy in proportion to the degree to which the nutrition of the patient is impaired. The temperature (taken in the rectum) is a good test of the gravity of the child's condition. If it remain at or above 105° F., or shoot up suddenly from a low to a high level, the danger is great; on the other hand, a fall in the temperature is a hopeful sign; and if the bodily heat become normal (in the rectum) we may look for recovery, although no immediate improvement be noted in the number or character of the stools. In addition to a high temperature other signs of serious import are:—persistence of the vomiting, heaviness and tendency to collapse, noisy breathing, convulsions, loss of elasticity in the skin. The last symptom is one which it is important to watch closely, for little improvement can be expected until the elasticity of the skin is restored.

C. **Choleraic or Septic Diarrhoea (Summer Diarrhoea: Cholera Infantum).**—This form of the illness is especially a complaint of hot weather and crowded cities. Although not unknown in later childhood, it is in infancy, during the cutting of the milk teeth, that the disorder is most prone to occur; and from its rapid course, its grave symptoms, and its too commonly fatal ending, is one of the most justly dreaded of the illnesses of early life. It is a noteworthy feature of the complaint that the patients are so often sturdy, well-nourished babies; for strong children are not less likely to suffer from it than weakly ones, and when seized may die almost as quickly.

Symptoms.—The complaint comes on quite suddenly. The child may have seemed quite well up to the time of the attack, or may have suffered for a few days from a slight ordinary looseness of the bowels. All at once, however, he is found to look pale, pinched, and ill. He vomits, throwing up first the food he has taken; afterwards a thin watery mucus coloured more or less deeply with bile. His motions, in the beginning thin, feculent, and very offensive, soon turn to mere serous fluid, which loses for the most part its repellent odour, and leaves only a yellowish stain with no trace of faecal matter upon the diaper. The action of the bowels is not attended by any griping or tenesmus, but the frequent profuse stools so drain the tissues of fluid that the child falls away with startling suddenness. After an absence of only a few hours we find him so changed that we hardly know him again. His eyes are sunken, his cheeks are hollow, and his nose is pinched and thin. His body, too, has shrunk up and his flesh feels soft and doughy. The skin is inelastic, especially over the abdomen: often it lies there in loose folds, and the abdominal wall may be as deeply hollowed as in the

most pronounced case of tuberculous meningitis. Thirst is of course extreme. If the child can talk he asks continually for drink. If he be an infant, he sucks his lips and lies whining fretfully with his eyes fixed upon any cup or vessel holding fluid. But his thirst is little relieved by what he takes; indeed, anything swallowed is usually returned in a few seconds. His lips are dry, and the wrinkle which curves on each side round the angle of the mouth makes them seem to project. The tongue, if clean and moist at first, soon becomes dry and brown. The urine is scanty and may be quite suppressed. The pulse is rapid and weak. The temperature is high. Even when the skin is cool to the touch and the feet are quite cold a thermometer held in the rectum will often register a heat of 104° or 105° F. On this account the temperature of the axilla or groin must not be taken as a guide to the internal heat of the body. At first the patient is very restless and cannot sleep; he throws his arms about and whimpers feebly as long as he has strength to do so; later, he lies in a drowsy state with dull eyes and half-closed lids, taking no notice of any one. His fontanelle is deeply sunken, and in a young infant the bones of the skull can be felt to overlap.

Vomiting now usually ceases, but small watery motions continue to be passed at intervals. Very soon the patient sinks into a state of collapse. He lies quietly on his back in his cot, his eyes shewing white between his half-closed purple lids. His face looks thin and pinched, and is wrinkled like the face of an old man. The complexion is earthy or lead-coloured, and his extremities, and even his nose, feel cold to the touch although the internal temperature is still high. In this state all reflex phenomena are abolished. The patient is insensible to all efforts to feed him, and seems unable to swallow; the sensitiveness of the conjunctiva is lost; the faint breathing alone shews him to be alive, and this soon comes to an end. A few feeble convulsive movements may precede death, and the temperature may rise to 106° or 107° F.

The course of the complaint is very short; it may last from a few hours to a few days. Almost always before the end of a week the child has either ceased to live or has turned the corner and begun to mend. If a favourable change occur we first notice a fall in the temperature; then vomiting ceases and the patient begins to retain fluids: he is less thirsty, and his motions become coloured again with faecal matter or bile. The looseness rarely ceases suddenly, but thin feculent stools continue to be passed, although less often and in smaller quantity. Sometimes the improvement in the stools is noticed before the vomiting has stopped, but the usual course is that described.

Diagnosis.—Choleraic diarrhoea is easily recognised. The intense thirst and obstinate vomiting, the sudden falling away as the tissues shrink up, the copious serous stools without a trace of faecal matter, and the early collapse form a very characteristic group of symptoms.

Prognosis.—The prospects of a patient stricken with this complaint are never otherwise than gloomy. In young babies especially the rate of mortality is very high. The danger is in proportion to the height of the

temperature, the urgency of the vomiting, the frequency of the stools, and the degree to which the elasticity of the skin is impaired. The last symptom is one which should always be carefully noted; even towards the beginning of the complaint, when the other symptoms, perhaps, do not denote any very pressing danger, if the skin is found to be inelastic we must look to the issue of the illness with grave misgivings. If the child become collapsed, the prospect is even more gloomy than in cases of ordinary inflammatory diarrhoea. In the latter complaint the patient may often be roused by energetic treatment; but in choleraic diarrhoea a favourable change is very difficult to establish, and if the patient be a very young child or infant recovery can hardly be anticipated. On the other hand, any abatement of the symptoms is to be welcomed as a valuable sign of improvement. Early cessation of the vomiting, a return of faecal matter to the stools, or, in particular, a fall in the internal temperature of the body, furnish solid ground for hope that the patient may eventually recover. But in infancy a favourable issue to the illness must always come to us rather as a surprise, especially as a good state of nutrition and previous sound health seem to influence the prognosis but little. Collapse may shew itself earlier in a weakly infant and later in a strong one, but in each case the end, when it comes, is apt to be the same. Fortunately in older children recovery is more common; and the outlook may be taken as less and less sombre in exact proportion to the advance in years.

Prevention of Diarrhoea.—In a large majority of cases the diarrhoeas of children may be looked upon as a consequence of want of knowledge or want of care. At least they may generally be prevented by attention to the feeding and management of the child, the cleanliness of his nursery, and the healthiness of his surroundings. This is especially the case in hot weather, when the worst kinds of bowel complaint are likely to be met with. To protect a child from diarrhoea we should put him into the best conditions for health. If he live in a crowded city he should be removed in the summer to an airy, well-drained house in some country place. Here he can spend his days out of doors, drink new milk, and sleep in a pure atmosphere. If he cannot be sent out of town we must do our best to keep him from bad air and stale or tainted food. The child should pass as much of his time as possible in the fresh air, spending a large part of the day in the parks, and sleeping at night in a well-ventilated room. The sanitary arrangements of the house must be carefully looked to: water-closets must be kept clean and regularly disinfected; attention must be paid to the housemaid's sink, which should not be used for the disposal of bedroom slops; soiled linen must never be allowed to remain in the nurseries; and care must be taken that the child's living and sleeping rooms are not near to an open drain-pipe, however thoroughly this may be trapped or cut off from the sewer. Many an attack of diarrhoea has been set up by a water-closet or housemaid's sink just outside the nursery door.

The child should be carefully guarded from chills. The washing of

his body must be a rapid process, and he cannot be allowed to prolong his bath unduly or play in the water. At night he should be lightly covered, and if he toss about in his sleep means must be taken to prevent his throwing his coverings aside and lying naked in his bed. The day dress is not to be neglected. Many young children suffer greatly from too scanty clothing, and especially from bare legs. Even in the summer the rapid changes of temperature inseparable from our climate are full of risk to an ill-protected child. If there be any suspected delicacy of constitution, or known susceptibility to colds, the legs should be covered with long woollen stockings, and the hips and thighs with woollen drawers.

In the matter of feeding we must see that all jugs, cups, feeding-bottles, and the like are kept clean, and that no food but such as is wholesome and in good condition is provided. It is best to boil or sterilise the milk without loss of time and put it aside in a refrigerator or some cool place. It must not be allowed to stand in a living-room. All meals must be freshly prepared, and it is unsafe to keep milk through the night for the early meal in the morning. Before the fresh milk is brought to the house it is wise to employ condensed milk or one of the desiccated milk foods.

In sultry weather the child, if thirsty, is not to be deprived of water; but this should be boiled and filtered. An infant may suck water from his feeding-bottle, and older children may be allowed to drink freely between their meals. Milk must be looked upon strictly as a food and not as fluid to be swallowed whenever the child is thirsty. The summer fruits should not be given to infants, but if ripe and sound and freshly gathered may be included in the dietary of older children. Unripe, stale, or tainted fruits must not be taken on any account. Meals should be fixed at regular intervals, and should not be too large. It is unwise to overload a child's stomach at any time, but in hot weather excess of saccharine or starchy food is especially liable to promote acidity and set up diarrhoea.

In the case of septic diarrhoea the contagious nature of the complaint must be recognised, and care be taken that infection is not conveyed from one child to another. In hospital wards especially, close attention must be paid to the thorough purification of the hands of the nurses who attend upon such cases. A nurse after changing soiled diapers must be made to cleanse and disinfect her hands with care before proceeding to other work; and it is wise to adopt the rule which I understand to be in force at the Babies' Hospital in New York, viz. that no nurse whose duties lie in handling the napkins of children so affected can be allowed to take any part in the distribution of food to the patients.

Treatment.—In the treatment of diarrhoea in the child we have to put a stop to the drain from the bowels, to lessen or remove the ill consequences arising from it, and to restore the normal processes of nutrition. If the complaint be a mere catarrhal derangement of little moment its management is an easy matter; but if the whole system have

been contaminated by the absorption from the bowel of the products of poisonous decompositions, our utmost ingenuity and resource may be taxed to guide the complaint to a favourable issue.

In the case of a mild catarrh of the intestine our first care must be to keep the child in an equable temperature and to avoid every source of chill. While the catarrh lasts the natural sensitiveness of the body to changes of temperature is heightened, so that the derangement once started may be prolonged from day to day by an exposure which in the beginning would have been powerless to set it up. For this reason the belly must be covered with a flannel binder, and while the purging lasts a general washing of the body must be forbidden, although for the sake of cleanliness rapid local sponging after a stool may be allowed.

If we have to do with an infant, and find him crying and drawing up his legs with abdominal pain, while the motions consist of undigested food and mucus, an aperient dose of castor oil or rhubarb and soda should be ordered at once to clear away the irritating matters. The laxative may be repeated if at any time undigested food be found in the stools, or the belly become hard and distended. After the action of the aperient the infant may take a few doses of the time-honoured chalk and catechu mixture. This is especially indicated when the stools are frothy and sour-smelling. If preferred, a few drops of tincture of rhubarb may be given, with half a drop of laudanum and a drop of sal volatile in an aromatic water. For diet the child, if not at the breast, should be fed with milk and freshly made barley-water in equal proportions, alkalinised with fifteen or twenty drops of the saccharated solution of lime.

This treatment will quickly put an end to an ordinary simple diarrhoea; but if the child continue to be bathed as usual, or be in any other way exposed to chill, the purging may resist the remedies, or return after apparent arrest. If this happen, three or four grains of rhubarb with half the quantity of aromatic chalk must be given at bedtime, and the dose may be repeated on the following night if the stools remain frothy and sour-smelling, In the daytime a twelve-months-old child may take oxide of zinc (gr. j.), or subnitrate of bismuth (gr. v.) and aromatic chalk (gr. j.) three times a day; or the rhubarb and sal volatile mixture as directed above. If, later, the stools become thin and watery, a little tinct. opii (♏ ss.-j.) can be added to the dose. Astringents are useful at this stage, and the opiate is of especial service. The treatment must be continued as described under the head of inflammatory diarrhoea.

In cases in which the catarrh occupies the lower bowel, and slimy stools are passed with straining efforts, small doses of castor oil and opium are indicated. Two drops of the oil with one of ipecacuanha wine and half a drop of laudanum may be given to a twelve months' infant three times a day. This form of looseness, and indeed any kind of simple diarrhoea, may be kept up by irritation low down in the rectum. Therefore in cases which do not yield readily to treatment it is advisable

to order a small injection (two drachms) of thin warm starch to be thrown into the bowel immediately after each motion.

Green stools, if acid or loaded with bile, require alkaline remedies. The green stool which is neutral, or only slightly acid, and gives no biliary reaction, can often be quickly changed for the better by a teaspoonful of a 2 per cent solution of lactic acid (as recommended by Hayem) given every four hours. In all cases soiled diapers should be removed from the room, and indeed from the neighbourhood of the nursery, without loss of time; if a green tint is noticed in the stools it is well to steep the napkins in a solution of corrosive sublimate (1 per 1000).

The looseness of the bowels which is common in teething infants must not be allowed to continue. Purging during dentition is in no way a beneficial process, but tends still further to reduce a child who is probably already depressed by pain and want of sleep. A weakly infant whose bowels are irritable and often relaxed may lose strength to an alarming degree after but a few days of the derangement.

After the age of infancy simple diarrhoea must be treated much in the same way as described above. While the derangement lasts the child should be confined to the house and made to discontinue his ordinary washing-bath. In the matter of diet he must avoid acid-making things, such as fruit and sweets. For medicine he should take an aperient dose of castor oil or rhubarb and magnesia; and when irritating matters have been cleared away he may begin an aromatic and antacid draught of sal volatile and spirit of chloroform with a drop or two of laudanum in cinnamon or peppermint water. This draught taken three times a day will soon put a stop to the disorder.

Lienteric diarrhoea is very amenable to treatment. For a child of five years old, one drop of Fowler's solution of arsenic and the same quantity of tincture of nux vomica may be given before meals three times a day, in a draught with dilute nitro-hydrochloric acid and infusion of calumba. At the same time the abdomen must be protected with a substantial flannel bandage; the feet must be kept warm; and every source of chill must be carefully guarded against.

In a case of *inflammatory diarrhoea* the patient should be put to bed at once. Even an infant or young child is better in his cot than resting on the lap of the nurse, heated by contact with her body and breathing her used air. The abdomen must be covered with a flannel binder, and the daily washing-bath must be put a stop to.

In the matter of *diet* it is of great importance for the time to give up the use of foods which are capable of decomposing to form poisonous ptomaines. Unless in the case of a breast-fed infant milk is to be forbidden; and even infants reared entirely on the breast will sometimes cease for the time to digest their mothers' milk. Bottle-feeding with milky foods is out of the question, however carefully the milk may be sterilised. Instead, the child should take freshly made whey diluted with an equal proportion of barley-water, or weak veal- or chicken-broth, mixed

with an equal quantity of fresh and fresh barley water. These foods may be given cold in alternate meals in small quantities at short intervals. If the infant be very weakly or shew signs of exhaustion, white wine whey should be given in doses of one tablespoonful as often as may seem desirable. If the weakness be great, the child may take no other food until his strength improves. As he begins to mend, Mellin's Food may be added to the meals, but milk is not to be resumed until convalescence is well advanced, and then only with great caution.

An older child can be fed with small meals of whey, barley-water, or broth diluted with barley-water. If the strength shew signs of failing, the brandy-and-egg mixture (B.P.) can be given. Still, in this matter of feeding we must remember that the weaker the child the more feeble his digestive power, and that food must be given in small quantities only. Strong jellies and extracts of meat if used must be well diluted with water, and every care must be taken not to overtax the feeble organs. As a rule, however, the patient is more eager for drink than for food, and at any age may be allowed to slake his thirst with cold water (boiled and filtered) as often as he wishes for it. Infants must be carefully watched for signs of thirst, and in any case notable shrinking of the tissues is a sign that water is urgently needed.

The air of the room must be kept fresh. In suitable weather the window must be opened, taking care that the child's cot is out of the direct line of draught. But few persons should be allowed in the room, and all soiled linen must be taken away at once.

In all cases of inflammatory diarrhoea the bodily heat must be carefully noted and reduced if it rise too high; it should be kept below 103° F. In taking steps to lower the temperature we must remember the tendency in this complaint to sudden collapse, and have stimulants at hand to correct any sudden failure of the heart. The temperature may be brought down by sponging with tepid water, by placing the patient in a bath of 80° F., or by enemas of cooled water. Of these methods I prefer the last if the child can bear it. The shock is in exact proportion to the coldness of the water, and therefore it is best to begin with the use of water cooled down to a temperature of 80° F., three or four ounces of which may be thrown into the bowel. If this be well borne the temperature of the enema may be slightly reduced on the next occasion. Ice-cold water has been recommended; but injections of this temperature must be used with great caution in a complaint in which the tendency to sudden collapse is so decided. Too rapid a reduction of the bodily heat may be attended with alarming symptoms of heart failure, and I have known a child's life to be brought into imminent danger by this means. Even at a temperature of 80° the injection has a markedly lowering effect upon some children, and we must be vigilant, therefore, to note any sign of depression. It is wise to administer a dose of brandy both before and after the operation, and to place a hot bottle wrapped in flannel against the child's feet when he is returned to his cot. If necessary a hypodermic injection of four or five drops of ether may be given.

If the elasticity of the skin be impaired we must take steps at once to restore its healthy action. To gain this end I know of no plan more useful than the wet pack. The child is wrapped as high as the armpits in a towel wrung out of cold water (made stimulating with a sixth part of eau-de-Cologne or brandy) and is then closely covered from the neck downwards with dry blankets well tucked in. The patient may be kept thus swaddled for six, eight, or ten hours, or longer; but every three hours he must be taken out, rubbed dry, and quickly repacked as before. A dose of spirits of nitrous ether (℥ x.-xx.) given in a little water every two hours during the process is a great help in this treatment. I look upon the state of the skin as due to faulty action of the kidneys, and have found nitrous ether quite without an equal as a quick and certain diuretic for young children. Besides helping to restore the normal suppleness of the skin the wet pack also tends to lessen the fever. In certain cases, however, the contrary effect is noted, and the bodily heat rises instead of falling. If this happen the wet pack must be abandoned as hurtful, and we must trust to the nitrous ether alone.

The use of *internal remedies* must go hand in hand with the other methods of treatment. As a first step, if the child be seen early, a dose of castor oil is always useful to clear away irritating matters from the bowel; but when this has been done we seldom find benefit from the ordinary astringents so long as the bodily temperature remains high. Although no definite rules for treatment have been arrived at as a result of bacteriological inquiry, some form of antisepsis of the bowel, if this can be attained, seems to offer the best prospect of success. With the exception of calomel, however, antiseptic remedies, it must be confessed, have not as yet given satisfactory results. I have made use of salol, β-naphthol, naphthalin, resorcin, iodine, carbolic acid, and the salicylates, but can boast of but little advantage from their employment. Given alone they have seemed to me to be almost inert. I have thought them of service only when combined with small doses of calomel or grey powder. After a fruitless trial of new remedies I have had again and again to fall back upon the old-fashioned combination of calomel and Dover's powder given in frequent small doses, and believe it to be the best form in which an intestinal antiseptic can be prescribed. In addition to the germicidal action of the mercurial, the opium is useful in regulating the extravagant peristalsis, and the ipecacuanha has a very beneficial action upon the mucous membrane of the bowel. I give a quarter of a grain of each every three hours to a one-year-old infant, and the improvement is often so striking and immediate that I cannot but attribute it to the action of the remedy. I have tried calomel in combination with most of the antiseptics already enumerated, and the perchloride of mercury with resorcin, but with less obvious benefit. Ipecacuanha has long been esteemed for its influence in improving disordered states of the intestinal mucous membrane, and is, indeed, as useful in derangements of the bowels as in catarrhs of the lungs. If the colon be the part affected,

or if there be excessive vomiting, ipecacuanha should on no account be left out of the prescription.

When the presence of mucus in the motions shews the lower bowel to be affected, medication by the rectum becomes useful. Irrigation of the bowel by warm saline solutions is of great service in these cases. In the East London Hospital for Children, with the help of the resident physician, Dr. R. P. Cockburn, I made large use of this method of treatment during the summer of 1896, and found that if injected slowly and gently, so as to throw in a large quantity of fluid, the irritability of the bowel was appreciably diminished, the stools were reduced in number and improved in quality, and, moreover, a certain absorption of fluid took place from the bowel, giving a perceptible increase of fulness to the frame. The strength of the solution used was one teaspoonful of common salt to the pint of warm water. The irritability of the lower bowel may also be lessened by the injection of small quantities of laudanum (℥ij.-iij.) or cocaine (gr. $\frac{1}{5}$) in two teaspoonfuls of thin warm starch after each motion; or by the use of suppositories containing the same quantity of sedative. When the rectum is acutely inflamed and prolapses like a bright red glistening ball which cannot be replaced, the protruded part must be bathed after each stool with warm water, and then covered with a thick poultice of boiled starch. Twice a day the laudanum and starch injection may be used with the addition of five grains of powdered ipecacuanha before applying the poultice. At the same time small doses of castor oil (℥ij.-iij.) should be given with half a drop of laudanum and one drop of ipecacuanha wine in a little aromatic water every four hours. Vinum ipecacuanhae in the same dose, but given every hour, will usually arrest the vomiting, which is often a distressing feature in these cases; or the same object may be gained by one-fifth of a grain of cocaine given several times in the day.

If prolapsus ani continue after the inflammation has been reduced, or come on later from relaxation of the sphincter and irritability of the mucous membrane of the rectum, the protruded bowel must be sponged and returned after each motion, and a small cocaine injection can be given as recommended above. If the mucous membrane become flabby as a result of repeated catarrhs of the bowel, an enema of infusion of rhatany or of nitrate of silver (gr. ij. in 4 ounces of water) used every night will usually put an end to the trouble, after a few repetitions.

When the fever has subsided the diarrhoea often becomes amenable to ordinary astringent remedies. Of these I prefer the extracts of haematoxylon (gr. j.-iij.) and rhatany (gr. j.-iij.) and the tincture of catechu (℥ v.-x.) to gallic acid or lead. Bismuth and aromatic chalk also form very useful remedies, and opium should always be added if there is any undue force in the expulsion of the stool. To be successful the dose of bismuth should be considerable, and as the action of the drug is purely local, large quantities may be given to quite young children without fear of ill consequences. I prefer the subnitrate to the carbonate, and give it in doses of not less than ten grains every two, three, or four hours, com-

bined with a grain of aromatic chalk and a small dose of resorcin or other antiseptic. If, as may happen, all astringents seem to irritate, the old prescription of dilute nitric acid and opium is very useful. For a child twelve months old two drops of dilute nitric acid may be given with half a drop of laudanum in water sweetened with glycerin three times a day. The same cases are often benefited by the sal volatile and laudanum mixture recommended for simple diarrhoea (*vide* p. 690).

When signs of prostration are noticed they must be attacked without loss of time. The child should be put into a hot mustard bath at once and held there for five minutes, or less if the blueness disappear earlier from his lips. Afterwards he is to be wrapped in flannel, and returned to his cot with hot bottles to his feet and sides. Every hour or half-hour a teaspoonful of the brandy-and-egg mixture, or white wine whey (according to the age), should be given; and, if exhaustion be great, a weak mustard and linseed-meal poultice can be applied for several hours to the chest and epigastrium. The mustard bath, although it will raise a lowered temperature, will not reduce the bodily heat when this is high; therefore the fever should be brought down by injections of cooled water as already recommended.

In the summer of 1896, with the assistance of Dr. Cockburn, at the East London Hospital for Children, I made trial of various cardiac stimulants in the hope of finding some effectual remedy to rouse the patient from his collapsed state. Of these camphor, which has been so highly recommended, seemed of little value unless given hypodermically with ether. The inhalation of oxygen was quite useless. The drug which produced the most immediate and striking effect was strychnine. The $\frac{1}{100}$ of a grain—that is, a quarter of a minim of the Pharmacopoeia solution—introduced under the skin seemed to give some life to the infant; but the effect only lasted about three-quarters of an hour, after which time the dose had to be repeated. We found that if given at shorter intervals a certain rigidity of muscles was produced by the remedy. In cases of pronounced collapse I believe this method of treatment to be a useful addition to our resources. When the patient is merely exhausted without being collapsed, a few drops of ether, given hypodermically, have a strongly stimulating effect. When the child is prostrate he must be kept in a horizontal position, and the nurse should be forbidden even to raise his head from the pillow.

In cases of recovery the mucous membrane is left in a relaxed state, and requires bracing up by tonic remedies. A few drops of the liquor ferri pernitratis are very useful at this time, given well diluted and in combination with tincture of nux vomica. During convalescence, and indeed for months afterwards, the susceptibility of the patient to fresh chills must be kept in mind, and he should be dressed warmly in woollen underclothing, and wear a flannel band to his belly.

The treatment of *choleraic diarrhoea* is greatly hampered by the obstinate vomiting, which not only forbids the stay of medicines on the stomach but baulks all our efforts to supply much-needed nourishment.

So long as this excessive gastric irritability continues it is best to hold over all attempts to feed the patient, and to content ourselves with giving iced water, in small quantities, as often as the child is willing to swallow it. The liquid is usually returned at once, but the thirst is so extreme that it is cruel not to do our best to satisfy it.

In all these cases the skin is excessively inelastic, and no time should be lost in packing the child in the wet towel as already recommended for inflammatory diarrhoea. If the vomiting be urgent, a poultice containing one part of mustard to five parts of linseed meal should be applied to the epigastrium before using the wet pack. In addition, one-sixth of a grain of calomel may be placed on the tongue every half-hour. Sometimes the vomiting stops after a few repetitions of this dose, especially if the stomach has been previously washed out. Instead of calomel other salts of mercury may be used, such as the perchloride or biniodide. Dr. A. P. Luff speaks favourably of the latter remedy in doses of gr. $\frac{1}{50}$, with 1 grain of chloral hydrate given every three hours to a child between the ages of six and twelve months. If other means fail, resorcin may be tried in doses of 2 or 3 grains every three hours. The stomach is sometimes quieted by a few doses of this remedy, but the drug has little or no influence upon the diarrhoea.

We must take advantage of any pause in the vomiting to supply food. This must be given at first in small quantities and in the simplest form. I prefer white wine whey, which combines food and stimulant, given from time to time in a teaspoon; but plain, freshly made whey, veal-broth and fresh barley-water or kumiss may be used, with the addition of five or more drops of pale brandy. The foods must be used cold, and if the quantities first allowed be kept down, may soon be given more liberally. Many children after some hours will take half a bottleful of white wine whey without harm at one meal; but any return of the vomiting should make us fall back at once upon the smaller quantities first recommended, or revert to cold sterilised water until the stomach is quiet again. Whatever kind of nourishment be chosen, it must be looked upon as food to be given at stated intervals, and not as drink to slake the thirst. For the latter purpose cold water is to be used, as already advised. If the patient become collapsed and the rectal temperature be high, the fever must be reduced by the means recommended for inflammatory diarrhoea. If cold enemas be employed, the temperature of the water at first should not be lower than 80° F., and stimulants must be given freely both before and after the use of the syringe. Hot bottles may be required in the cot if the fall of temperature after the injection is considerable, and the hot mustard bath or even hypodermic injections of ether may be necessary. In addition to their influence in reducing temperature, the rectal injections are of service in supplying a certain amount of fluid to the tissues. I have tried hypodermic injections of the saline solution with the same object, and have found that 2 ounces injected into the loose tissue of the back were absorbed slowly, and seemed to give a little relief; still, so long as the exhausting discharges from the bowels continue, the small

amount of fluid which can be introduced by the skin can do little to satisfy the wants of the system.

As long as the temperature remains high the purging must be regarded as a beneficial process tending to prevent accumulation of noxious organisms in the body, and no attempt should be made to interfere with it. But when the fever has subsided special measures may be taken to bring the looseness more under control, and any of the remedies recommended for inflammatory diarrhoea may be made use of. I have had good results from the subnitrate of bismuth, but the quantity given must not be less than 10 grains every three or four hours to a child of twelve months. I usually combine each dose with 2 grains of salicylate of soda and half a drop or so of laudanum.

If the patient be seen early, a hypodermic injection of morphia will often cause an immediate improvement in the more alarming symptoms, and moderate the vomiting and purging at once, or even completely arrest it. This, in children of four or five years old and upwards whose strength enables them to withstand the enfeebling influence of the complaint, may be sufficient to determine a favourable issue. Infants and the younger children, however, have not this reserve power and are more easily depressed. In them, therefore, although the irritability of the stomach and bowels may be held in check, the course of the illness is often not materially improved, and the patient sinks into a state of collapse and dies just as if the vomiting and diarrhoea had not been interfered with. The chances of recovery are much greater if the remedy can be adopted quite early in the illness. If it be delayed until exhaustion sets in, it may seem merely to add to the weakness of the patient. One thirty-fifth to one-fortieth of a grain may be used for an infant twelve months old combined with five drops of ether; and the dose may be repeated in two or three hours if a sufficient effect have not been produced. It is better to administer the narcotic in small doses at short intervals than to begin with a considerable dose at first, for in the beginning it is impossible to tell how susceptible a young child may be to the action of the remedy. If the child become collapsed, stimulating hypodermic injections of strychnine must be used as directed for inflammatory diarrhoea.

Chronic Diarrhoea.—Chronic diarrhoea is a very dangerous and insidious form of disease which yields reluctantly to treatment and is often fatal to infants. It may be the sequel of an acute attack, or come on by slow degrees in a state of health. In the younger subjects the complaint is usually at first a mere catarrhal derangement. It may be so likewise in older children, but in the latter it is often the result of tuberculous ulceration of the bowel.

Etiology.—In infancy most cases of persistent diarrhoea are the consequence of chilling of the surface. The leisurely way in which the washing of an infant is often carried out, without any reference to the state of his health, is no doubt answerable for many illnesses; and

an intestinal catarrh thus induced may be kept up from day to day and week to week by continually recurring impressions of cold. A child who lives in this state of catarrh almost always has a languid circulation; and his cold feet and legs—exposed as they are too often to every change of temperature—make him keenly susceptible to chill.

In the same way an intestinal catarrh set up by the continual worry of the mucous membrane by fermenting food may be prolonged almost indefinitely by a persistence in the same diet. This is well seen in cases in which feeding with milk and starch is not given up. There are few cases of chronic diarrhoea in the child which do anything but badly upon a milky or farinaceous diet. Such foods supply material for the fermentative action of bacteria. The microbes attack the milk-sugar and carbohydrates generally, and give rise, as Escherich has pointed out, to the formation of acetic and lactic acids, carbonic acid and hydrogen. From investigations carried out at the Rockefeller Institute for Medical Research it appears that the more virulent organisms are absent from the stools of children who suffer from this chronic form of the derangement even when they contain mucus in considerable quantity.

In older children chronic diarrhoea is frequently the consequence of cold feet or careless exposure in the bath. It is sometimes also a sequel of the infectious fevers and whooping-cough; but in these cases, too, I believe the exciting cause to be chill acting upon a system rendered more than commonly susceptible by the late illness. In strumous and tuberculous children a chronic diarrhoea may be kept up by tuberculous ulceration of the bowels.

Symptoms.—When the chronic complaint follows an attack of acute diarrhoea the child, although the more urgent symptoms have subsided, remains weak and pale; his temperature is normal or even lower than in health; his appetite is poor; and his bowels discharge several times a day a thin, dark, offensive fluid or sour-smelling pasty matter with mucus.

It is more common, however, for the disorder to begin gradually. The patient—usually a child of fifteen or eighteen months—is noticed to be looking pale and dull. His flesh is flabby and his feet are cold. His spirits, however, keep good, his temperature is normal, and he sleeps well at night. Soon he is noticed to be obviously thinner, and his strength begins to fail so that his love of movement leaves him; he is never happy out of his nurse's arms, and, if previously able to walk, may refuse to stand or even to rest his weight upon his feet when held to the ground. This change in his habit excites a good deal of comment and some anxiety, but the child's appetite continues good and his bowels —the nurse will declare—are "nicely open." In all such cases the stools should be inspected, and it will be found that some two, three, or more evacuations occur in the twenty-four hours, and that these are large, often putty-like, and always offensive. The nurse will insist that the child passes more than he takes. Certainly the greater part of the food he swallows is discharged undigested from the bowels.

These symptoms go on for weeks without change. The patient con-

tinues to lose flesh and seems to grow duller and paler every day. His bowels are variable. Sometimes he may have only one or two copious pasty stools. At other times the evacuations are more numerous and thinner or even watery. At times, indeed, there is actual diarrhoea, and for a few days the patient is seriously purged, the stools being frothy and sour-smelling, or thin and dark coloured like dirty water. They are always exceedingly offensive. As the complaint goes on the attacks of acute diarrhoea return more and more frequently until the bowels become persistently loose. Still, it may be months before the stage of actual diarrhoea is reached. For a long time the child, although weakly, ailing, and spiritless, has a good appetite; indeed, often he shews great eagerness for his food. He is not feverish; on the contrary, the internal temperature of the body is often below the normal level, and his extremities are always cold. He perspires copiously at night about the body, sleeps uneasily, and is very difficult to please. There is, however, no pain; and the absence of more definite symptoms in a child whose nutrition is so obviously at fault may be a cause of much perplexity.

When diarrhoea is established the stools vary in character from time to time. They always have a horribly offensive smell and are dark coloured and watery, or drab coloured like thin paste, or very loose like chopped spinach diffused through a brownish water. In advanced cases they deposit a shreddy sediment mixed with small black clots of blood. We may then suspect ulceration of the bowel, especially if there be any tenderness of the abdomen on deep pressure.

The number of stools is subject to great variety. At times the purging abates for a time. The bowels are then moved only two or three times in the day, and the child is brighter and more easily amused. The looseness then returns, and the patient is relaxed ten or a dozen times in the twenty-four hours and again becomes dull and spiritless. The persistent diarrhoea interferes seriously with the child's nutrition, and the loss of flesh goes on quickly. The face gets hollow and lined; the eyes are dull, sunken, and tearless; the forehead is wrinkled, and a curious look of age is given by a deep furrow which passes from the nose round the corner of the mouth. The complexion is earthy, especially about the forehead, and the expression is peevish or sad. The fontanelle is deeply depressed. The abdomen is often swollen from flatulent distension, but the liver and spleen are of normal size, and no enlargement can be felt in the mesenteric glands. In this stage the perspirations usually cease, and the skin over the whole body becomes harsh and dry. The coldness of the extremities is remarkable, and the hands and feet may have a purple look; often they are swollen from oedema. The water is scanty and may contain lithates or a little uric-acid sand. The child is very weak and lies quietly in his cot, taking little notice of anything. If able to talk he often refuses to answer questions, or replies merely by a whine or a fretful shake of the head. His appetite may still be good, but often it is poor, so that the child can only be fed with

difficulty. In this state the patient may lie for weeks, getting more and more feeble, and die at last gradually from pure exhaustion or suddenly from collapse of the lung. Sometimes death is preceded by the symptoms of "spurious hydrocephalus."

Although chronic diarrhœa in the infant usually occurs during the period of dentition, it does not necessarily bear any special relation to the appearance of the teeth. Often the teeth are cut regularly and easily without affecting the progress of the diarrhœa either for good or ill. In other cases—and this is seen especially at the later period of dentition—the coming of each tooth is marked by a notable increase in the purging, and the looseness moderates at once when the tooth breaks through the gum. That a catarrhal derangement should be influenced by teething is no matter for surprise. When the gums are turgid and inflamed there is always some fever, and a feverish child is necessarily more sensitive than another to the ordinary causes of chill.

The frequent variations which take place in the number of the stools and the severity of the symptoms is very distressing to those interested in the patient, for their hopes are being continually raised up only to be thrown down again. After a time, however, with care, a real improvement begins, and the first sign of amendment is a change in the appearance of the stools which grow more homogeneous and solid and begin to contain bile. The child looks brighter in the face, and when he cries the eyes again become suffused and watery. This reappearance of tears is a sign of improvement of no little value. The fetor of the stools is slow to subside. Even after the child has gained considerably in flesh and strength the stools often remain exceedingly offensive. Recovery, too, is never uninterrupted. Relapses almost always occur, however carefully the child may be tended and fed. These often follow a fall in the barometer: indeed, the sensitiveness to barometric changes is so extreme that on damp days the patient is always less well than on days which are bright and dry.

In children of four or five years of age and upwards a chronic diarrhœa is more likely than in an infant to be dependent upon some constitutional cachexia. I have known a syphilitic ulcer of the intestine to keep up a looseness for many months before the nature of the lesion was detected, and in consumptive families there is often tuberculous ulceration of the bowels. In the latter case the abdominal swelling is accompanied by signs of tenderness on pressure; there is fever; the superficial veins of the abdomen are often visible; the liver is perhaps enlarged from fatty change, and swollen mesenteric glands can often be detected on deep pressure in front of the spine. The nutrition of the child necessarily suffers, and he gets pale and thin, but the rapidity of the wasting depends less upon the ulceration than upon the violence of the purging, the amount of fever, and the presence or absence of disease elsewhere. The prognosis, however, is serious, for the intestinal complaint is almost always only one of several manifestations of the constitutional mischief.

After recovery from a prolonged attack of chronic diarrhoea the patient remains for many months anaemic, weakly, and subject to relapse. The digestion is very poor and any—even the smallest—error in diet may be followed by a return of the derangement. This is true not only of infants, but also of children of seven or eight years of age and upwards. In the latter an occasional consequence of the complaint which ought to be mentioned is a curious check to the growth of the patient. I have seen several such cases. In one of these—a little girl who suffered for five years from repeated and prolonged attacks of chronic diarrhoea—the child, who in 1897 was ten years old, weighed 26 lbs. 9½ oz. and measured only 3 ft. 3¾ in. in height. During the year 1896 she had grown only ⅞ in. Another little girl also suffered for several years from repeated attacks of chronic intestinal catarrh, each of some months' duration and separated by periods of comparative health. She had, I believe, at one time ulceration of the bowel. This child, when 8½ years of age, measured 3 ft. 6⅛ in. and weighed 2 st. 12 lbs. Both the patients were curiously small for their ages, and were only kept in good health by strict diet and constant care.

Diagnosis.—In every wasting infant the motions should be inspected as a matter of course. If this be done the nature of the complaint can hardly be overlooked. Examination of the stools is of especial importance in the early stage before diarrhoea has become confirmed. At this time the mother makes no complaint about the bowels: indeed, she rarely refers to them except to say that they are not relaxed. Careful inquiry is therefore necessary to determine the fact that of the food taken the larger part is escaping digestion altogether. When this point is settled, and we notice the normal evening temperature, the cause of the loss of flesh is no longer a mystery. Any fear, therefore, that the child is becoming the subject of tuberculosis may be laid aside.

It must be remembered, however, that teething gives rise to fever, and that the complaint is most common during the period of the first dentition, so that a febrile temperature may complicate a non-febrile disorder. In the case of infants this source of fallacy must be kept in mind, and every examination, to be complete, must include a careful inspection of the gums. Still, with regard to tuberculosis in young children, it may be laid down as a rule that, so long as there is a sufficient cause, such as obvious digestive derangement, to account for the wasting, we are not warranted in going beyond what we see and in suspecting tuberculosis or anything else in the background. In very many instances I have had occasion to congratulate myself upon observing this very reasonable precept.

When the diarrhoea has become constant, ulceration of the bowels may be suspected if there is moderate increase of tension of the abdominal wall, especially in the right iliac fossa, or sign of tenderness on deep pressure. The stool most characteristic of the lesion consists of an offensive dirty brown liquid containing scraps or flakes of solid matter and little black blood-clots. In infants ulceration, if it occur, is rarely

due to constitutional causes, but in older children it may be of a tuberculous nature. The chest, therefore, should be examined for signs of consolidation and the abdomen for any indication of peritonitis. If these can be excluded and the evening temperature be normal, we have no grounds for suspecting the existence of constitutional mischief.

Prognosis.—So long as the purging is only occasional the child under appropriate treatment may be expected to do well. It is in cases in which the looseness has become a confirmed diarrhoea that the issue is doubtful. The danger is greater in infants, and in them is serious in proportion to the degree to which general nutrition is impaired. The appearance of thrush in the mouth is a bad sign; and oedema of the extremities, as it shews marked poorness of the blood, warns us that the strength of the patient is getting low. Ulceration of the bowels in an infant is a very serious condition, and likely to prove fatal. In older children the patient may still recover if well treated and nursed, provided that the lesion be not dependent upon diathetic influence. Even in the latter case I have known recovery to take place in a strumous subject in whom from the nature of the symptoms and the character of the stools I believed tuberculous ulceration of the bowels to be present.

Treatment.—To succeed in the treatment of chronic diarrhoea, we must look upon the complaint as consisting of a series of catarrhs of the bowel due in the main to separate impressions of cold. We must take every care, therefore, to guard the sensitive body from chill, and to provide a diet which will not overtax the weakened powers of the stomach or supply material for bacterial fermentation.

In the early stage of the complaint, when the stools consist of masses of pale, pasty matter, it is best to forbid milk in any form, and to use instead fresh whey and barley-water mixed in equal proportions with a good spoonful of Mellin's food. If the patient be an infant he may take for other meals fresh whey and cream, weak veal-broth and barley-water, or the yolk of an egg beaten up with whey or veal-broth. A sufficient variety should always be prescribed, and care must be taken that the whey and such-like foods are in good condition, for stale whey or old barley-water is sure to disagree. The child's feet and legs must be swathed if necessary in cotton-wool, and the washing-bath must be carried out in such a way as not to chill the child.

For medicine, a few grains of prepared rhubarb may be given at night with a grain or two of aromatic chalk powder, and in the daytime half a drop of laudanum three times a day in an alkaline mixture with carminatives. This treatment will soon alter the character of the stools, and if due care be taken to protect the child from cold, milk may soon be resumed; but it is always wise to give one of the desiccated milk foods or other form of preserved milk for a few days before beginning the fresh cow's milk. As a help to digestion Finkler's papain is very useful given in doses of two grains with each bottle of food.

In the case of older children, besides forbidding milk, we must restrict the quantity of farinaceous matter. Toast or malted bread and

rusks are to be preferred to ordinary baker's bread; and well-boiled vegetables, such as cauliflower, or vegetable marrow, should be substituted for potatoes. Eggs are harmless, and mutton, poultry, game, and white fish may be taken freely. Variety should be aimed at, and it is wise to provide with each meal some form of nitrogenous food, such as meat, egg, bacon, or thinly sliced ham or tongue, so that the patient may not be dependent upon toast or rusk for the means of satisfying his hunger.

If in spite of this diet the stools still continue to be pasty and large, the amount of starch should be still further reduced. A welcome help in such cases is raw or very underdone mutton pounded and rubbed through a fine wire sieve. Of this food the child should be encouraged to eat largely, and often he takes it with great relish.

At the same time that the diet is regulated, the stools must be carefully watched. If they become loose, the treatment should be that recommended for the acute attack. Offensive stools require antiseptic remedies, such as resorcin, naphthalin, or perchloride of mercury; and with each dose it is well to combine a drop or two of laudanum to quiet too energetic a peristaltic action of the bowels. Excess of mucus in the evacuations is usually stopped by the castor oil and opium mixture recommended for certain cases of inflammatory diarrhoea. In very chronic cases, in which the motions are habitually frequent and watery, nitrate of silver should always be given in doses of gr. $\frac{1}{8}$-$\frac{1}{5}$ with a drop or two of laudanum and the same quantity of dilute nitric acid three times a day. In many cases we find that the patient improves as long as the nitrate is being taken, and falls back at once when the remedy is discontinued. I have had many such cases under my care, and have been forced to persevere with the silver medicine for months together in order to ensure a favourable ending to the illness. There is no danger in this continued use of the drug, although I have known a fear to be entertained by some physicians as to a risk of pigmentation of the skin by the metal if it be taken by a young child for more than a few weeks together. I believe this apprehension to be quite baseless, for I have found that it requires very many months of uninterrupted use before any such result is to be expected. I have given the drug continuously for eighteen months to young children without producing any such consequence, and believe that it might be taken without a break for a still longer period without risk of harm.

Cases of chronic diarrhoea can usually be cured if care be taken to keep the patient warm and to protect him from every source of chill. Until he is well advanced towards recovery the washing-bath should be viewed with great suspicion. When washing is needed the patient should be placed in hot soap-suds and sponged rapidly, and no soaping outside the bath should be allowed. In bad cases, however, it is wiser, while the bowels remain loose, to forbid all washing of the child's body, with the exception of local sponging after the stools, and to keep his feet, legs, and belly thickly swathed in cotton-wool. I have in my mind not a few

patients who owed their recovery, in my opinion, mainly to the use of this simple precaution.

EUSTACE SMITH.

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E. S.

INTESTINAL OBSTRUCTION

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PART I.—THE GENERAL CONSIDERATION OF INTESTINAL OBSTRUCTION

SYNOPSIS:—1. Definition. 2. The General Pathology of Intestinal Obstruction. (A) Morbid Anatomy—(i.) of a Case of Chronic Obstruction; (ii.) of a Case of Chronic Obstruction which has become Acute; (iii.) of a Case of Acute Obstruction of Normal Intestine. (B) The General Consideration of the Symptoms of Intestinal Obstruction and their Pathological Explanation. 3. The General Symptoms of Intestinal Obstruction—its Three Clinical Varieties. 4. The Diagnosis of Intestinal Obstruction—(i.) the General Diagnosis; (ii.) the Differential Diagnosis of Different Causes; (iii.) the Differential Diagnosis from Similar Conditions. 5. Terminations, Duration, and Prognosis. 6. The General Treatment.

1. **Definitions.**—Intestinal obstruction is a condition in which the intestinal contents are more or less hindered in their passage along the bowel. The mechanical and organic causes only of obstruction of the intestinal lumen will be dealt with here, as the dynamic causes of obstruction, such as paralysis and spasm of the intestine, are referred to elsewhere.

Ileus is an ancient term incapable of precise definition, and therefore

should not be used. It is often employed to signify paralytic dilatation of the intestine, but has been applied to any form of abdominal distension, and is sometimes used as synonymous with intestinal obstruction. *Stenosis* is a narrowing of the lumen of the intestine. *Occlusion* is complete closure of the lumen of the intestine. *Obturation* of the intestine is that condition in which the lumen is blocked by a foreign body. *Stricture* is stenosis or occlusion of the lumen of the gut from some disease attacking an annular portion of its wall. *Constriction* is stenosis or occlusion of the intestinal lumen by some cause surrounding the gut. *Strangulation* refers to the condition of a coil of intestine when it is so constricted that its lumen is occluded, its vessels are obstructed so that, as a rule, intense venous hyperaemia is produced, and its nerves are vigorously excited.

2. The General Pathology of Intestinal Obstruction. — (A) **Morbid Anatomy.** — Intestinal obstruction has three distinct clinical forms—(i.) Acute intestinal obstruction in which the normal intestine is suddenly and completely occluded, and, as a rule, some segment of the gut is strangulated. In such a case the changes in the strangled loop are perhaps the most important, but other changes are to be found in the segments of intestine above and below the obstruction. These changes are for the most part the result of the interruption of the intestinal stream, but so rapidly fatal are these cases that little time is allowed for structural alteration. (ii.) Chronic intestinal obstruction. There are many causes for this condition, but all agree in that the lumen of the intestine is narrowed so that a partial obstruction is produced. The prolonged course of these cases allows well-marked anatomical changes to occur above the obstruction. (iii.) Cases in which the symptoms of acute obstruction supervene on those of a chronic stenosis; the anatomical changes are very similar to those in the preceding class, but some, such as distension and accumulation of faecal matter above the obstruction, are emphasised. In discussing the morbid anatomy of intestinal obstruction it will be convenient to describe, first, the changes found in chronic obstruction, then those of chronic cases which have terminated acutely, and, lastly, the lesions found in acute intestinal obstruction.

(i.) **Morbid Anatomy of a Case of Chronic Obstruction.**—We are not concerned here with the causes of the chronic and progressive stenosis, which will be described below.

The intestine below the obstructed point is pale, contracted, and, as a rule, empty, and like the intestine of a starving animal. In a few cases, however, of stenosis in the small intestine, and especially when opium has been freely administered, a considerable bulk of faeces may collect in the colon. Thus, in a case of simple stricture of the small intestine under my care the diagnosis was at first somewhat obscured by a large accumulation of hard faeces in the sigmoid flexure and rectum.

The changes in the segment of intestine above a stenosis are well marked and characteristic. They are distension, elongation, hypertrophy, inflammation, and ulceration of the mucous membrane, and in some cases

striation of the mesentery from stretching. For a variable distance and degree the coils of intestine are dilated above every stenosis. But narrowing of the gut does not invariably obstruct the passage of the intestinal contents sufficiently to produce these changes. As the contents of the small intestine are habitually fluid or gaseous a very considerable degree of narrowing may exist without offering any obstruction to the passage of the contents; while in the lower part of the colon, where the contents are solid, a much less degree of narrowing will produce pathological changes. In the early stages of a progressive stenosis, dilatation is confined to the coils immediately above the obstruction, and is only moderate in degree. When the lumen of the bowel has been nearly occluded, and especially when from any cause acute and total obstruction has supervened, the dilatation and distension may be enormous, and the whole of the intestinal canal above the stenosis may be affected. The contents of these dilated coils in the lesser degrees of distension are those normally found in the part of the alimentary canal involved. In the small intestine and in the upper part of the colon they will be fluid, but in the sigmoid flexure and the rectum large masses of solid faeces may accumulate above the obstruction, but even in the lower colon in advanced degrees of stenosis the contents are, as a rule, liquid. As the obstruction becomes more complete, and these contents stagnate and ferment, an irritative secretion from the intestinal wall is added to them. As long as the circulation is not seriously interfered with, gas will not accumulate in the distended coils, for it is absorbed by the vessels of the intestinal wall. Moreover, gas always passes through the stenosis more readily than fluid faeces. The distended intestines necessarily increase in length; this mechanical result of distension of an elastic tube is illustrated by the varicosity seen in veins and arteries submitted to excessive pressures. The greatly distended and lengthened intestines are thrown into folds, as is particularly well shewn in volvulus of the sigmoid flexure, in which, on opening the abdomen, the gigantic coils of distended colon are found folded upon themselves and often occupying the whole of the abdomen.

Striation and shortening of the mesentery.—One of the functions of a mesentery is to provide a certain amount of slack peritoneum, so that as the coil of intestine becomes distended it may spread between the layers of the mesentery or mesocolon without hindrance. In the excessive distension above a stenosis the mesocolon is often shortened by this means almost to the point of disappearance, and it may therefore be exceedingly difficult to bring a loop of distended sigmoid flexure out of a colotomy wound. In rare instances the whole of the slack peritoneum provided by the mesentery or mesocolon is utilised, and the strain thus thrown upon the peritoneum produces striae in the line of the axis of the gut.

The factors leading to hypertrophy of the intestine above a stenosis are in part mechanical distension and in part irritation of the mucous membrane above the stenosis by the accumulated contents of the intestine. Patel asserts that hypertrophy does not occur in the absence of

ulceration; in support of his contention that ulceration rather than obstruction is the determining factor, he points out that in intestinal obstruction due to pressure from without there is distension without hypertrophy, and, as is well known, that extensive ulceration without organic obstruction will produce well-marked hypertrophy. In the latter case, however, spasmodic stricture is probably present during life. Nothnagel investigated the nature of this hypertrophy in man, and his pupil Herczel by experiments on animals. The hypertrophy of the intestine is due to an increase in the muscular tissue and a dilatation of the smaller blood-vessels supplying the muscular coat. The connective tissue undergoes no change; the muscle-fibres are not increased in number, but become larger, and more especially broader, the hypertrophy being manifest by the fifth day, and well marked by the ninth day, after the production of experimental stenosis. The degree and extent of the hypertrophy depend upon the amount of the stenosis and the time occupied in its production, hypertrophy being always best marked immediately above the stricture.

Inflammation and ulceration of the mucous membrane above a stenosis.—Enteritis nearly always exists above a fully developed stricture, and in most cases numerous large shallow ulcers are present. These stercoral ulcers present every transition from chronic indolent forms to sloughing and gangrenous patches. A group of such ulcers is found usually just above the stenosis, but others are scattered irregularly along the distended portion of the intestine. In obstruction of the colon there is often a group of characteristic ulcers in the caecum, an event of importance, inasmuch as peritonitis frequently originates from these ulcers, either by the transudation of micro-organisms or by a gross perforation. These stercoral ulcers are seldom due to the irritation of hard impacted faecal masses. They are the result of interference with the circulation due to the distension combined with the chemical irritation of the intestinal contents and bacterial infection. Kocher has suggested that "Distension Ulcers" would be a better name.

(ii.) **The Morbid Anatomy of a Case of Intestinal Obstruction which has become Acute.**—The causes of such a sudden transition are numerous, and will be narrated later (*vide* p. 725), but for purposes of illustration it may be stated here that a foreign body, such as a plum-stone, may become impacted in a stricture, or a coil of distended intestine may be kinked or twisted, or the exhausted intestine may suddenly become paralysed and relaxed. The morbid changes found in such a case will differ in degree only from those that have been already recounted. The distension and elongation will be far more extensive. The rapid accumulation of food, secretions, and gas in the last few days of life will distend such an alimentary canal from the stenosis to the stomach. The sudden rise of intra-abdominal tension will interfere with the circulation, and the gases will no longer be absorbed from the intestinal lumen, and will rapidly increase the distension, thus establishing a vicious circle. The shortness of the time will not permit of any increase of hypertrophy, but

the enteritis and ulceration of the mucous membrane above the obstruction will be greatly increased, chiefly as a result of the sudden distension. Peritonitis from perforation or from the passage of micro-organisms through the coats of the intestine follows, frequently almost immediately after the obstruction becomes acute.

(iii.) **Morbid Anatomy of a Case of Acute Obstruction of Normal Intestine.**—In nearly all forms of acute intestinal obstruction not only is the lumen of normal intestine suddenly and completely interrupted, but a coil of intestine is strangled in addition. In some cases a band is knotted or noosed around it, in others the intestine passes through a hernial ring or an aperture in some membranous structure such as the mesentery or omentum ; in some axial rotation takes place, and a volvulus is produced ; and in others, again, a part of the intestine is invaginated within a part below and gripped by it. In whichever of these ways the acute obstruction is produced, three different conditions are shewn by (*a*) the intestine below the obstruction, (*b*) the intestine above the obstruction, and (*c*) the strangled coil within the obstruction. An exception to this general rule is when a large gall-stone or foreign body passes suddenly into the normal intestine and abruptly interrupts its lumen. In these cases the first two conditions only of the intestine occur.

The changes found in acute intestinal obstruction are very different from those already described in a chronic stenosis. Hypertrophy and chronic inflammatory changes of the mucous membrane are entirely absent from the coils of intestine above the obstruction, but distension is, as a rule, even more marked in this portion of the gut. On the other hand, the changes in the strangled coil are profound and very evident, and the results of these changes on the general organism through the nervous and vascular systems produce the predominant symptoms of the disease.

(*a*) *The Morbid Anatomy of the Intestine below an Acute Obstruction.*—These coils are empty, contracted, and pale, and in the case of the small intestine no thicker than the little finger. No doubt at the moment of obstruction their normal contents are present, but when seen a day or two later at an operation or autopsy the coils are completely empty. This condition is probably brought about in the manner described in Dr. Bayliss and Prof. Starling's experiments upon animals. The first effect of resisting the progress of intestinal contents, or of pinching or otherwise stimulating the intestine, is powerfully to augment the peristaltic waves above the stimulated point, and to paralyse and relax the intestine for three feet below it. In this way the intestine endeavours to overcome the obstruction and force on its contents. This augmentation of peristalsis is, however, evanescent, and soon the waves of peristalsis pass over the point of stimulation or obstruction and continue on along the intestine, which then becomes contracted and empty, and apparently passes into a condition of spasm. The condition of the intestine below a strangulation is therefore rather one of emptiness and spasm than of paralysis, as is so often stated.

(*b*) *The Morbid Anatomy of the Intestine above an Acute Obstruction of*

Normal Intestine.—As has already been said, these coils of intestine do not present hypertrophy or ulceration. They are, however, greatly distended. Their walls are pale and thin when the obstruction is recent and very sudden; when it is of longer duration they are thickened by oedema and deeply congested with blood, but they are never the same dark purple colour as the strangled loop. Distension and dilatation are the most evident changes in the intestine above an acute obstruction. This distension may be local or diffuse. In many cases, and probably in all early cases, it is confined to the intestine immediately above the obstruction. In cases of some days' duration it is diffused, and the whole of the alimentary canal is dilated as high as the duodenum and the stomach. The contents of these dilated coils are for the most part liquid and pultaceous, and to only a small degree gaseous. Gas does not tend to accumulate until the circulation of blood through the intestines is so much interfered with that absorption ceases. This occurs, however, when general peritonitis complicates an acute obstruction. The degree of the distension of any coil of intestine is ultimately limited by the resistance of its own wall and the pressure exerted on it by the abdominal wall and adjacent coils of intestine. The generally accepted explanation of the origin of this distension is stasis of the intestinal contents. These contents consist not only of the food taken, which in such cases is very little, but also of the secretion of the various digestive glands. The secretion of saliva is on an average one or two pints a day, and each of the other glands probably supplies a similar amount. In acute intestinal obstruction with vomiting probably a continuous and excessive secretion is produced by reflex irritation. The alimentary canal may roughly be divided into an upper part, consisting of the mouth, stomach, and duodenum, devoted to secretion, and a lower part, the colon, in which most of the water is abstracted from the intestinal contents. Nearly all forms of acute obstruction affect the small intestine, and in this way the area of secretion is shut off from the area of absorption. As a result the coils of intestine are flooded and distended by the secretion of the digestive glands, rendered still more excessive by the reflex irritation of the stagnating contents. The flood rises higher and higher from one coil of intestine to another until the duodenum and stomach are submerged, and it overflows in the gushing vomiting of the later stages of acute intestinal obstruction. The question of the faecal character of this vomit is considered on p. 714. When the obstruction is in the colon, as in acute volvulus of the sigmoid flexure, the distension of the gut above the occlusion is much less marked, for here a part of the area of absorption is still available. It has been urged by some that a diffuse paralysis of the intestine, due to the lesion producing the obstruction, plays a part in the production of the dilatation of the intestine, and no doubt the toxic contents paralyse and relax the intestinal wall in the same way as nicotine and cocaine have been shewn to do experimentally, but no amount of paralysis or relaxation would produce dilatation unless the contents accumulated or were increased by excessive secretion.

(*c*) *The Morbid Anatomy of the Strangled Coil of Intestine.*—A coil of intestine may be strangled in any of five ways: a band-like structure may encircle its root and mesentery; it may pass through an orifice into a hernial sac, in which case the ring-like orifice acts in the same way as a band; a loop of gut may work its way through an aperture in the mesentery or omentum, the margins of the orifice producing strangulation; one portion of intestine may become invaginated into the coil below it, which tightly grips the intussuscepted portion and its mesentery, especially around its neck; or lastly, a coil of intestine may, by axial rotation, twist the entering and leaving coils so tightly around the base of its mesentery as to obstruct the venous return and strangle the coil.

In whichever of these ways strangulation of a coil of intestine is produced the results are the same. The pre-eminent consequences are vascular engorgement and violent stimulation of the sympathetic nerves supplying the coil. As a secondary result of interference with its circulation the coil becomes distended to its utmost capacity with gas and with effused blood, and lastly the lumen of the alimentary canal is interrupted completely and suddenly by the mechanical pressure and by the paralysis of the strangled coil of intestine. The subject of strangulation of intestine has been investigated experimentally by Kader, whose results are summarised by Nothnagel. He placed ligatures of various tensions around coils of gut in animals, and observed the result. He also ligatured the mesenteric vessels separately and in combination with ligatures, occluding the gut at one point like an annular stricture.

The Vascular Changes in a Strangled Coil of Intestine.—When, for example, a band nooses a coil of intestine tightly, the veins returning from that coil are compressed and become distended and engorged with venous blood. The arteries continue to force blood into the capillaries of the coil until they are filled, at a pressure approaching that of the arteries, and the whole coil is distended and erect with venous blood. Within an hour or so the coil becomes deep purple in colour, tense, oedematous, and exudes a blood-stained serum into its lumen and into the neighbouring peritoneum. This blood-stained fluid collects in the peritoneal cavity, or in the hernial sac if one be present. In very rare instances the strangulation is so tight as to occlude the arteries at once; the veins are not distended with blood, and the coil of intestine dies and becomes gangrenous and flaccid in an hour or less. In such cases there is scarcely any distension of the strangulated coil. In other instances, which are still rare, the veins are occluded or nearly occluded at once, and the circulation ceases. In these cases distension, engorgement, and extravasation of blood occur, but the intestine dies in a few hours, the vessels become thrombosed, the arterial pressure is cut off from the coil, which exudes its serum and gas, and becomes flaccid, green, and gangrenous. In the vast majority of cases the obstruction of the veins is not complete, and enough blood escapes from the veins of the coil to enable a slow and greatly diminished circulation to be maintained, and for this reason such coils of intestine will often recover when released after three or four days

of strangulation. It cannot be too strongly urged that coils of gut are not dead and gangrenous because they are purple or black. The great majority will readily recover when their veins are released. The prevalent notion that black gut is always gangrenous is responsible for many unnecessary enterectomies. True gangrene of intestine is shewn by a green or grey colour combined with flaccidity and the presence of free gas and a stench in the peritoneum around the dead coil.

The Meteorism of a Strangulated Coil of Intestine.—When a coil of gut is moderately strangled its lumen becomes distended to its utmost capacity with blood and with gas. This gas is probably carbonic acid, and is produced by the vital processes of the tissues. It is unable to escape as it should by the obstructed veins. The same phenomenon is always seen when intestinal clamps are applied to a coil of intestine in performing gastro-enterostomy or intestinal anastomosis. The coil of intestine is distended to many times its normal extent, and the tension so exerted draws into the constricting ring fresh coils of gut, which are in turn strangled and distended. These coils draw their mesentery along with them within the constriction, which in this way becomes tighter and tighter in a vicious circle. The extent and proportion of the abdominal distension and meteorism due to the strangled coil depend entirely upon its size and accessibility to palpation. Where it is small and inaccessible there will be no distension due to this cause, as when a mere knuckle of gut passes into a hernial sac or orifice or is strangled by a band. Where, however, a large coil of intestine is affected, as in volvulus of the sigmoid flexure, the abdominal distension which rapidly supervenes is almost entirely due to the strangled intestine, and may soon assume gigantic proportions. The tension of the strangled coils is, moreover, much greater than that of the coils distended by stasis above the obstruction. It is well to remember that strangled coils within the abdomen are as tense as an ordinary strangulated hernia.

The Infection of the Strangled Loop of Intestine and of the Peritoneum.—Even before the coil of gut is dead and gangrenous it loses its power of preventing the passage of micro-organisms from its lumen through its wall to the peritoneum, and as a consequence the tissues are infected and peritonitis is set up which may spread to the whole peritoneum. Even if the strangulation be relieved at this stage the patient frequently dies of this infection and the inflammation of the intestinal wall and peritoneum, which cannot be removed.

Thrombosis, Gangrene, and Perforation of the Strangled Intestine.—Ultimately the vessels become thrombosed and the gut gangrenous in all unrelieved cases. The gases and blood escape by exudation and the strangulated coil is flaccid and green, whilst gas is found in the peritoneum and the exudate is stinking. In some cases crude perforations are present, but in many the orifices through which the gas and fluid contents of the bowel escape are microscopical. In a strangled coil of intestine, the capillaries, being unsupported and totally unprepared for

the strain, rupture, and haemorrhage occurs into the substance of the bowel and blood escapes into the neighbouring cavities.

A strangled coil of gut is paralysed. This is probably a combined result of the distension, vascular interference, and the direct injury to nerves in the mesentery by the cause of strangulation. The lumen of the intestine is usually doubly occluded at the points where the intestine enters and leaves the constriction. Moreover, the high tension in the coil and its paralysis would form an effectual block to the passage of intestinal contents even when the entering coil is not mechanically closed as sometimes happens in hernial orifices and apertures.

B. **The General Consideration of the Symptoms of Intestinal Obstruction and their Pathological Explanation.**—The pathology of intestinal obstruction includes not only the morbid changes produced, but the resulting functional disturbances. Under the present heading these disturbances and their explanation will be discussed in a general manner.

Pain.—(*a*) *Pain in Acute Intestinal Obstruction.*—Three varieties of pain are found when the intestine is strangled. The first pain is usually sudden in its onset and intense, doubling the patient up in his agony. It originates in the strangled coil, and is due to the mechanical injury of the constriction and to the stretching and distension of the coil by the venous engorgement and the gaseous accumulation. It is as a rule referred to the umbilicus in strangulation of the small intestines. This is a special instance of the general rule that sensations can only be localised in the segments of the alimentary canal which are more or less fixed, namely, the stomach, duodenum, and colon. Sensations from the small intestines, which wander freely about the abdomen, are experienced as diffuse pains, and are referred to the umbilicus. Thus, as I have pointed out, when a large gall-stone passes into the duodenum the pain is at first correctly localised to the epigastrium, but when the calculus has reached the small intestine the pain is diffuse and is referred to the umbilicus. Similarly, in volvulus of the sigmoid flexure or of the ileo-caecal junction the pain is as a rule correctly localised in the iliac fossae, and the same applies to intussusception or impaction of a foreign body at the ileo-caecal junction. In rare instances the pain has been definitely localised in very various parts of the abdomen although the lesion was in the small intestine, but for these cases no explanation has been offered. This first pain is continuous, so that the patient has not even momentary relief, until after a time the nerves of the strangled coil become exhausted and poisoned and the pain abates. It disappears entirely just before death, and in some of these cases gangrene of the intestine has been found ; in others a perforation has allowed the coil to collapse, and in others the remission of pain must be ascribed to the narcosis of septicaemia and profound collapse. In exceptional cases the pain has been insignificant from start to finish. Some of these patients have been very old ; in others the strangulation does not appear to have been abrupt in onset. This pain originates in the sympathetic nerves and is charac-

terised, as such pains usually are, by the association with vomiting, collapse, and profound vascular disturbance. It is more or less proportional to the extent of intestine implicated in the strangulation and to the severity of the constriction. The higher up in the intestine the lesion is situated the more severe as a rule is the pain; strangulation of the small intestine is always more painful than that of the colon, probably because the vagi take a larger share in the formation of the solar and mesenteric than in that of the hypogastric and inferior mesenteric plexuses. This explains why strangulation by bands and internal hernias, which as a rule implicate the small intestine, are much more painful than volvulus which is usually found in the colon.

The second pain is of the nature of colic. It is due to the tetanic and violent peristaltic efforts of the intestine above the obstruction to force the contents past the obstacle. These paroxysms of colic do not last very long, and become less and less severe and frequent until they stop. Their cessation is accounted for partly by the rule that no hollow muscular organ, whether intestine, bladder, or uterus, will struggle long with an insuperable obstacle; partly by the distension which thins out the muscular wall and places it at a great mechanical disadvantage, and partly by the poisoning of the neuro-muscular apparatus by the toxins formed in the stagnant contents. As a consequence of the last two causes the gut is frequently paralysed in long-standing cases, and is unable to empty itself even when the obstruction is relieved.

The third pain is stabbing, localised, and associated with tenderness and rigidity, and often with some rise of temperature. Unlike the first and second pains, it is not relieved but is increased by pressure which indeed cannot be borne. It is due to the onset of peritonitis and infection, and appears from the third to the fifth day, when micro-organisms begin to pass from the lumen of the intestine through the wall to the peritoneum.

(*b*) *In Chronic Intestinal Obstruction.*—The pain is usually of the peristaltic variety only. The lesion producing the obstruction is as a rule almost painless, and there is no intestinal strangulation. Peritonitis frequently appears at the end of cases of stenosis, but it is far from constant and is to be regarded as a complication. The onset of pain in this form of obstruction is as a rule gradual. At first it only gives rise to periods of discomfort associated with the rumbling of wind which "will not pass downwards." Gradually, as the stenosis becomes more complete and the coils of intestine above it more distended and hypertrophied, definite attacks of colic occur, and these become more frequent and severe. In these attacks the patient often cries out, is doubled up with the pain, often grasps his abdomen, and endeavours to rub away the lump formed by the tetanically contracted intestine. In other instances handling the abdomen appears to excite attacks, and is avoided by the patient. In obstruction of the colon the pain is often localised correctly by the patient, who is able to trace the peristaltic wave along the course of the large intestine with ever-increasing tension and distension to a climax of tetanic spasm at the stricture. The intestine can often

be seen to rise up as a writhing, rigid mass at the height of the spasm.

Vomiting.—(*a*) *Vomiting in acute intestinal obstruction* commences from half an hour to twenty-four hours after the onset of the pain, usually after a short interval. To some extent its onset and degree are determined by the severity of the pain, the presence or absence of food in the stomach, and the sensibility of the patient. Some people, especially children, vomit readily, others with great difficulty or hardly at all. The early vomiting of acute intestinal obstruction is associated with intense nausea, straining, and eructations, and gives no relief to the patient's sufferings. At first the vomited matter consists of gastric contents, but as time goes on it becomes green and bilious. The violence of the vomiting may to some extent lessen after a day or two. Finally, the character of the vomiting alters and it becomes gushing; the patient at frequent intervals opens his mouth, from the corner of which, without effort or straining, pours a foul liquid stream. The vomit has, as a rule, at this stage become faecal; it is entirely liquid, brown in colour, and of a horrible odour more or less resembling that of faeces. It greatly exceeds in quantity any fluids which may have been taken. It is at this stage that "drowning from faecal vomiting" is apt to occur when an anaesthetic is administered. As the patient becomes anaesthetised the sensibility and contractility of the pharynx, larynx, and oesophagus are annulled; suddenly the patient stops breathing, and a filthy liquid stream commences to pour in overwhelming quantity from his nose and mouth. Unless the head and neck be immediately lowered and turned upon the side, and the mouth forced open and the lower cheek depressed by a finger passed into it, the patient inhales the foul fluid into his lungs and either dies at once, or, if his lungs are rapidly cleared, a day or two later, of septic bronchopneumonia.

The early vomiting of acute strangulation is a reflex phenomenon of the sympathetic nervous system, and resembles that seen in biliary and renal colic, in crushing of a testis, in torsion of a testis or ovary, or when the peritoneum is violently stimulated by an acute infection or injury. The regurgitation of bile into the stomach during the second stage of vomiting has not been satisfactorily explained. It would appear that there is a greatly increased flow of bile into the duodenum, and if this be admitted, then, as Pawlow has shewn, the pyloric sphincter would be relaxed by the alkalinity of the bile. Even then it is hard to see why it enters the stomach in any quantity, unless antiperistalsis exists in the upper part of the alimentary canal. John Hunter, in his paper on intussusception, maintained that antiperistalsis occurred during vomiting, and for this reason recommended emetics as a desperate means of undoing intussusceptions.

Gushing and faecal vomiting are due to the stasis and accumulation of intestinal contents above the obstruction (*vide* p. 709). The profuse secretion of the salivary, gastric, biliary, pancreatic, and intestinal glands floods the intestines above the occlusion, distends the

stomach, often to an extreme degree, and finally overflows from the mouth. This explanation of gushing faecal vomiting was, according to Leichtenstern, first propounded by Huguenot of Montpellier (1713). It was later advocated by van Swieten and Henle. In support of this explanation it may be pointed out that the degree and early onset of gushing vomiting are practically proportional to the proximity of the intestinal occlusion to the stomach. When the obstruction is low down in the colon, not only is a much longer portion of the intestine available to contain the secretion, but the major part of the "area of absorption" is above the obstruction, and the water can be abstracted from the intestinal contents in a normal manner. Thus, in gall-stone intestinal obstruction in which the obstruction is at first in the duodenum the vomiting at the very start is gushing and copious, but abates as the calculus moves farther and farther down the canal. On the other hand, in volvulus of the sigmoid flexure gushing vomiting is often entirely absent.

The fundamental error, which has vitiated the whole problem of faecal vomiting and has produced a superabundant literature, is that the foul matter smelling like faeces must have been produced in the lower segments of the intestine and have been conveyed backwards to the stomach. At first it was supposed that this was true faecal matter, and that its presence proved that the obstruction was in the colon. This error was long ago exposed by the observations of morbid anatomy, but modern writers, such as Nothnagel, still think it necessary to explain the transportation of intestinal contents from a position immediately above the occlusion to the stomach. There does not, however, appear to be any evidence that this transference occurs, and it is therefore needless to explain it. It sometimes happens that innumerable fruit-stones accumulate above an intestinal stricture, and many such cases have terminated acutely with faecal vomiting, but the stones have always been found massed in the ampulla above the stricture and not distributed up the intestine. The same remark applies to metallic mercury, which in former times was given to overcome the obstruction.

The chief hypotheses to account for the regurgitation of intestinal contents from the lower segments of the intestine to the stomach may, however, be stated briefly. The oldest and most persistent explanation—that of antiperistalsis (motus antiperistalticus)—originally advanced by Galen was supported by John Hunter, and in modern times by Nothnagel, although with reservations. The experimental researches of Dr. Bayliss and Prof. Starling have shewn that when Auerbach's and Meissner's plexuses are paralysed short rapid waves pass indifferently up or down the intestine. These waves are purely muscular in origin and have very slight propulsive force. True peristalsis is a neuro-muscular act, and requires for its production the integrity of the whole muscular and nervous apparatus of the intestine. Dr. Bayliss and Prof. Starling do not state that they ever succeeded in reversing true peristalsis in their experiments; but W. B. Cannon, by incorporating bismuth subnitrate in

the food and enemas given to cats, was able to observe by *x*-rays and the fluorescent screen that the commonest movement of the ascending and transverse colon was one of antiperistalsis in this animal; large enemas were even driven through the ileo-caecal valve into the lower coils of the ileum. According to Nothnagel, a few indubitable cases have been recorded of the vomiting of formed faeces even before the physician's eyes, as in the cases narrated by Rosenheim, Desnos, and Schloffer. Some years ago a hysterical woman, under the care of Sir F. Treves, not only vomited true faecal masses, but enemas containing castor oil and subsequently methylene blue. Laparotomy was performed on this woman on no less than three occasions by different surgeons to cure a gastro-colic fistula, which was, however, shewn to be absent at each operation. Antiperistalsis undoubtedly exists in very rare cases, but as has already been pointed out it is not necessary to invoke it in the explanation of faecal vomiting.

Brinton advanced the hypothesis of a regurgitant axial current. He asserted that the ordinary waves of peristalsis in an occluded coil of intestine produce a forward movement of the contents along the sides and a reversed current up the centre of the gut. It has been proved, however, that the distended intestine is soon exhausted, and to all intents and purposes immobile as regards contractions. Others have suggested that the pressure of the diaphragm and abdominal muscles, especially during the act of vomiting, would empty the distended lower coils into the empty coils and stomach above them. It has been urged that since the coils cannot be emptied in a downward direction the contents must be forced upwards. This explanation, though plausible, is for mechanical reasons unsound; for in the abdominal cavity the upper coils would be submitted to the same rise of pressure as the distended ones, and no transference of contents would take place; to bring about this result the pressure would have to be concentrated on the distended coils.

Faecal vomit is of a brownish-yellow, brownish-green, or yellowish-grey colour, and has an offensive odour comparable to that of faeces. No chemical analysis of faecal vomit has apparently ever been made, but the colour is probably due to bile pigments reduced to urobilin by the decomposition going on in the fluid. In part it may be due to altered blood-pigment exuded from the congested and inflamed mucous membranes. The faecal odour is due to the products of the bacterial decomposition of proteins, which requires considerable time and in normal circumstances is not complete until the contents of the bowel have reached the lower part of the colon. Should the intestinal canal be obstructed so that the contents remain in the duodenum and stomach for a day or two, this decomposition is then completed there and a faecal odour is produced, nor is there any need to invoke antiperistalsis or a regurgitant axial stream to transport the decomposing matter from just above the point obstructed.

(*b*) *Vomiting in chronic intestinal obstruction* is not so urgent, per-

sistent, or severe as in strangulation of the intestine. As a rule it occurs at intervals, and then after food only. The patient, as a rule, discovers that so long as he is strictly moderate in the quantity of food taken and for the most part restricts himself to liquids he can control the vomiting. Sometimes vomiting occurs during severe attacks of colic. It is then comparable to the vomiting seen in any other form of colic and characteristic of all sympathetic pains. In several cases of cancerous stricture of the lower part of the large intestine with great distension of the transverse colon it has appeared to me that the copious vomiting which occurred at intervals would bear the following explanation:—The commencement of the transverse colon crosses the second part of the duodenum at right angles without the interposition of peritoneum or any mesentery, and when greatly distended must press upon the duodenum at this point and more or less obstruct it. The stomach was distended in these cases, and it appeared probable that this was due to a secondary mechanical obstruction produced in this way and without the intervening small intestine having as yet become fully dilated. This explanation would accord with the intermittent and irregular character of the vomiting and with the observation that it is sometimes very large in quantity, as in pyloric obstruction, and is always induced by a full meal.

Collapse.—In acute intestinal obstruction collapse is one of the earliest manifestations, but in the chronic form it does not occur until the final and closing stage, when the obstruction has become acute. In collapse the patient is prostrated with weakness, he is anxious and restless, the pulse is rapid, small, and thready, the temperature subnormal, and the skin and extremities cold and clammy with sweat.

Collapse in acute intestinal obstruction is the result of three causes, any one of which can produce collapse, but in acute intestinal obstruction they are combined until the profound and terrible collapse which immediately precedes death results. These causes are:—(1) An acute lesion of the terminations of the splanchnic and pneumogastric nerves in the abdomen; (2) depletion of the fluids of the body by the profuse vomiting and sweating, which produce a concentration of the blood; (3) intoxication and infection by the decomposing products, arrested by the obstruction, and by bacterial invasion. All three of these causes play a part at various stages in the production of collapse in acute strangulation of the intestine. In the terminal collapse of chronic obstruction the third is the predominant cause and to a less degree the second, whilst the first takes scarcely any part. (1) The reflex nervous factor has been fully investigated experimentally by Bezold, Goltz, Bernstein, and Ludwig and Asp. The first effect of a violent stimulation of the vagus in the abdomen is to slow the pulse, and this is sometimes seen in the early stages of any acute abdominal lesion. The subsequent result is paralysis of the cardio-inhibitory centre, and the pulse becomes rapid. Severe lesions of the vaso-constrictor branches of the splanchnic nerves at first produce a transient constriction of the vessels and a rise of blood-pressure, which in turn slows the rate of the heart. This rise

soon passes off and the abdominal arterioles are paralysed and the viscera become engorged with blood, the blood-pressure falls, and the heart beats faster. The superficial vessels are depleted of blood, the radial and other accessible arteries are small, and the pulse thin, thready, and weak. The skin is pale and cold, especially in the extremities—the hands, feet, nose, and ears. The cerebral circulation is small and precarious, and to this must be attributed the anxiety, fear, and restlessness. The cerebral anaemia also reduces the tone of the cardio-inhibitory centre, and in this way quickens the pulse. The centre which controls sweating is also interfered with, and the patient is bathed in a cold, profuse perspiration. Probably the thermogenic centres are also more or less paralysed, for, as Leichtenstern has pointed out, not only are surface temperatures depressed, but those taken in the rectum. (2) The depletion of water by vomiting and perspiration. In acute intestinal obstruction the patient frequently vomits pints of fluid in addition to that which he loses by profuse sweating and passes as urine. Any fluid drunk is often rejected at once by the stomach, and the attempt to take liquids by the mouth is abandoned. As a result there is rapid loss of weight, the blood is concentrated and becomes more viscous, and probably all the tissues of the body are similarly depleted. The most obvious sign of this rapid depletion of fluids is the falling-in of the cheeks and eyes. The latter become "ringed" and the whole face is pinched. The tongue is dry, small, and wrinkled, the secretion of saliva stops, the skin is wrinkled, and the urinary excretion is diminished or even suppressed. Whilst this is mainly due to the shortage of fluids in the body, the great fall of blood-pressure no doubt also plays a part, especially in the failure of the glomerular excretion of the kidney. At one time it was held erroneously that the diminution of urine was due to only a smaller part of the intestine being available for absorption, and therefore that the degree of anuria was an indication as to how high up in the alimentary canal the obstruction was placed. This is true only in so far that vomiting and collapse are proportional to the proximity of the obstruction to the stomach, and it is these factors which diminish the excretion of urine. According to Nothnagel, the concentration of the blood may reach 24 per cent of its volume, and the percentage of haemoglobin is correspondingly increased. This is no doubt an important factor in reducing still further an already weakened circulation, and, as in severe cholera, is probably responsible for muscular cramps, especially in the calves, and for such unusual and rare cerebral symptoms in intestinal obstruction as coma, delirium, and convulsions. (3) Intoxication and infection from the obstructed bowel. In the final phase of acute intestinal obstruction or of chronic obstruction which has become acute and absolute the patient presents a picture of profound poisoning. He lies prostrate and collapsed in bed, wasted to a grey gaunt shadow, with sunken eyes and fingers wandering aimlessly over the bedclothes. The skin is covered with a clammy sweat, and his parched, cracked tongue can produce nothing but a hoarse whisper. His pulse is uncountable and his

breathing irregular, sighing and flickering as though about to cease for ever. Yet he seems to suffer little pain, only to be haunted by a nameless dread. This final stage of collapse is due to the absorption of poisons from the obstructed coils, and less frequently to septicaemia. The list of poisons is extensive, but it is uncertain which, if any, produce these symptoms of sapraemia. These poisons are the products of the decomposition of proteins in the stagnant contents. Indol, phenol, skatol, and aromatic oxy-acids, hydrogen disulphide, acetone, and diacetic acid have all been credited with a share in the production of this condition. Others have regarded the poison as an alkaloid, or the product of the bacillus of malignant oedema or of the colon bacillus. Some special symptoms may be attributed to this terminal sapraemia and septicaemia, such as delirium, coma, pyrexia not due to peritonitis, and nephritis when it occurs.

In conclusion, it may be pointed out that collapse due to reflex nervous causes is abolished in an hour or so by morphine, and that that due to depletion of fluids can be rapidly met by saline transfusions, so that these two causes together need offer no insurmountable obstacle to recovery after the cause of the obstruction has been removed. On the other hand, when the collapse of obstruction is due to sapraemia or septicaemia, the most successful operation will usually fail to save the patient.

The Condition of the Bowels in Intestinal Obstruction.—In partial obstruction a diminished quantity of faeces and flatus passes through to the intestine below, and the faeces frequently accumulate for some time in the colon before they excite the desire to defecate. The flatus is voided daily, and an enema will at any time readily empty the colon of the faeces and flatus it may contain. This condition is known as one of "irregular and partial constipation." When the mucous membrane of the coils above the stenosis is inflamed and ulcerated, frequent fluid motions may be passed containing blood and mucus. This condition is one of "irritative diarrhoea." When acute strangulation has taken place the bowels are as a rule in a condition of "absolute constipation." Neither faeces nor flatus are voided, nor has the patient any desire to defecate. If an enema be administered to such a person it is often retained or is returned with little force and without result in flatus or faeces, except perhaps such as may be washed mechanically from the rectum. A motion is sometimes passed immediately after the onset of the pain and vomiting. On the other hand, one may be voided just before death and after days of absolute constipation. The condition of the bowel below a strangulation appears to be a most curious one. It has been customary to speak of it as paralysed, but when seen at operations or autopsies it is empty, contracted, and pale, and not relaxed and flaccid. The condition is rather one of inhibitory spasm. When a motion is passed soon after the onset of the attack, it is probably in consequence of this spasm being established. When one is passed just before death, it is because asphyxia has stimulated peristalsis. In some cases the voided matter is supposed

to have come from above the obstruction either by means of a perforation or owing to a relaxation of the parts at the moment of death. In acute strangulation of the colon tenesmus is frequently present. Thus, in intussusception situated at the ileo-caecal valve frequent small motions of blood and slime are passed. Tenesmus and straining are not uncommon in volvulus of the sigmoid flexure or in acute obstruction of a cancerous stricture of the colon. In very rare instances a strangulated hernia has been associated with profuse diarrhoea ("Choléra Herniaire," Malgaigne). Diarrhoea is even rarer in internal strangulation. The fluid is supposed to be a profuse secretion from below the obstruction, due to paralysis of the mesenteric nerves. Some of these cases have so closely resembled cholera as to be mistaken for it.

Indicanuria.—Indol is produced chiefly in the small intestine by the bacterial decomposition of proteins. It is excreted in the urine as indican (potassium indoxyl-sulphate). In acute obstruction of the small intestine its amount may rise to 100 to 150 milligrams daily (Jaffé), the normal quantity being from 5 to 20 mg. This increase may be used in the differential diagnosis between obstruction in the small intestine and in the colon. When indicanuria is well marked on the second or third day of an obstruction the occlusion is in the small intestine. When it is absent after several days of subacute obstruction the lesion is in the large intestine. Its presence in such a case, however, does not exclude a lesion of the large intestine, for the distension may have spread into the small intestine. Indicanuria also occurs in peritonitis, especially when diffuse, in tuberculous and typhoid ulceration of the small intestine, and in simple enteritis and cholera. It is also found in the chronic decomposition of pus in cavities, especially the pleural cavity.

3. The General Symptoms of Intestinal Obstruction.—**Its Three Clinical Varieties.**—The three clinical varieties of intestinal obstruction are:—(*a*) *Acute intestinal obstruction*; (*b*) *chronic intestinal obstruction*; (*c*) *cases of chronic intestinal obstruction which have terminated acutely.* These three forms of intestinal obstruction present the same symptoms, but in a very different degree and variety. The classical symptoms of intestinal obstruction in their usual order of incidence are pain, vomiting, collapse, constipation, and the various changes which may be observed on examining the abdomen. The following classification summarises the differences presented by these symptoms in the three clinical varieties:—

[Table

	Pain.	Vomiting.	Collapse.	Constipation.	Abdomen.
(*a*) Acute intestinal obstruction.	Sudden, severe, continuous, stabbing.	In half an hour, severe, continuous, gushing, faecal.	Early and profound. Temp. subnormal. Cramps. Thirst.	Absolute, and to enemas.	Usually normal at first.
(*b*) Chronic intestinal obstruction.	Gradual, intermittent. Colicky, with flatulence.	Late, only after food, seldom faecal.	Late, slight. Temp. normal.	Partial, alternating with diarrhoea, blood and slime. Morning diarrhoea.	Distended, visible contracting coils of gut.
(*c*) Chronic obstruction become acute.	1. The symptoms of (*a*) supervene on a history of (*b*). 2. Onset less sudden and severe than in (*a*). 3. Abdomen distended and visible contracting coils of gut. 4. History of previous attacks cured by enemas and purges.				

Pain is the earliest symptom in all forms of intestinal obstruction. In acute intestinal obstruction the patient, who may be in perfect health, is suddenly struck down by the most severe abdominal pain without any warning. The pain is so exceedingly severe that it doubles him up, is continuous, and never remits. At first it is stabbing, and is due to the actual damage to nerve-endings in the intestine and peritoneum. The second pain comes on in exacerbations of the first and is of a colicky character. The third variety of pain does not supervene until the strangled coil has become inflamed and peritonitis has commenced, and it is therefore associated with tenderness on palpation (*vide* p. 712). The pain in chronic intestinal obstruction forms a marked contrast to the above. The patient has been in ill-health for some considerable time, suffering from flatulence, dyspepsia, and more or less abdominal unrest, and he has perhaps wasted. The pain gradually becomes worse during twenty-four or thirty-six hours, until the patient has severe attacks of colic; but these occur only at intervals, and between the attacks he is free from pain. These attacks of pain are due to the peristaltic and tetanic contractions of the hypertrophied intestine above the stenosis.

Vomiting in acute intestinal obstruction usually comes on half an hour after the pain, but it may be delayed for twelve hours. It is characterised by nausea and severe retching, and continues without intermission, becoming worse and worse until it is faecal in character from the second to the seventh day. The vomit at first consists of gastric contents, then becomes bilious, and finally is a fluid faecal stream

of gushing vomit, which nothing will arrest except a large dose of opium. In chronic intestinal obstruction the vomiting does not occur until the third or fourth day and is never a prominent symptom of the disease. As long as the patient abstains from food and is quiet he does not vomit, although he may feel sick. Gushing faecal vomiting is seldom seen, and then only when the case has terminated acutely. The contrast between the vomiting in acute and in chronic intestinal obstruction may be summed up as follows: The higher up in the alimentary canal the cause of the obstruction is the more severe, profuse, and continuous will the vomiting be and the less the distension. On the other hand, the lower the obstruction is in the intestinal tract the greater will be the distension and the less and later will the vomiting be. Acute obstruction as a rule implicates the small intestine; chronic obstruction usually the colon.

Collapse is the third symptom to appear in acute intestinal obstruction, and it is usually well marked in a few hours. The patient is pale, cold, and sweating. The hands and feet and the tips of the nose and ears are cold and clammy. The pulse is a feeble thread or perhaps impalpable. At first it may be slow, but soon its rate is increased. The temperature is subnormal. The patient suffers from prostrating weakness, dreadful thirst, and often from severe cramps in the calves of the legs. The final toxic stage of collapse has been described on p. 718. In chronic intestinal obstruction, on the contrary, the collapse is not often severe until late in the disease, when the patient is distended and poisoned by the products of the bacterial decomposition in his alimentary canal. The temperature is as a rule normal or slightly raised.

Constipation in acute intestinal obstruction is as a rule absolute. It is true that the bowel may empty itself immediately after the onset of the pain and vomiting, but after that neither faeces nor flatus are passed, nor has the patient any desire to defecate. The best diagnostic method of testing for the presence of absolute constipation is the double turpentine enema. Two enemas, containing half an ounce or an ounce of turpentine in half a pint of soap and water, should be given with an hour's interval. The first enema is intended to wash away such faecal matter as may be contained in the rectum. The second is, however, the enema upon which attention should be concentrated, and three points should be carefully noted concerning its return: first, the amount of faecal matter and bile evacuated; secondly, whether flatus is returned with and after the enema, and for this reason care must be exercised that air is not injected with the enema; thirdly, whether the enema is returned with force, or merely flows away, or is retained, for this is a gauge of the degree of inhibition of the colon. In acute intestinal obstruction the second turpentine enema is retained or flows back without much force and brings away neither faeces nor flatus. The exceptions to this general rule are the tenesmus, which occurs in intussusception and sometimes in volvulus of the sigmoid flexure, and the very rare form of diarrhoea sometimes associated with any form of strangulation.

The constipation in chronic intestinal obstruction presents several curious characters. It is incomplete and irregular, the patient passes flatus at all times and faeces at irregular intervals, and a turpentine enema will always readily empty the rectum of its contents of flatus and faeces. Frequently diarrhoea alternates with constipation, or scybala are passed in a fluid stool. Sometimes there is a passage of blood mixed with slime. One of the earliest signs of an ulcerating cancerous growth low down in the colon is "morning diarrhoea," that is to say, as soon as the patient rises in the morning he is obliged to pass a fluid motion. If this sign appear in a man over 40 who has had no previous abdominal trouble, and especially if it be associated with flatulence, the rectum should be examined and the abdomen palpated for a carcinoma of the colon.

The abdominal examination in the early stages of acute intestinal obstruction as a rule reveals very little. The abdomen is usually flat, not distended, rigid, or tender. In intussusception and volvulus there are, however, well-marked signs. An intussusception can be felt under an anaesthetic in about three-quarters of the cases as a sausage-shaped, tender tumour which hardens and softens. A volvulus will greatly distend the abdomen and render it extremely tender quite early in the disease.

In chronic intestinal obstruction the abdomen is distended, frequently to an excessive degree. The most certain sign, however, of chronic obstruction is visible peristalsis. When in a case of stenosis of the intestine the light is arranged so as to strike transversely across the abdomen, and the latter is gently kneaded, coils of intestine will very frequently be seen to rise up and harden in vermicular contractions, and waves of peristalsis will be seen to pass from one part of the abdomen to another. These movements are usually associated with borborygmi. The condition of intestine revealed by this sign can only have been produced by a partial intestinal obstruction of some one or two months' duration. This obstruction has produced hypertrophy and distension of the coils of gut above the stenosis, and at the same time emaciation has thinned the abdominal wall. These three factors combine to render the waves of peristalsis visible. In extreme emaciation the normal intestine may be seen in a state of peristalsis through the abdominal wall, but the coils do not harden as they contract.

Cases of Chronic Intestinal Obstruction which have terminated acutely.—In the third clinical variety of intestinal obstruction the symptoms of acute obstruction supervene on a history of chronic obstruction. The history is usually somewhat as follows:—The patient is, as a rule, over 40 years of age, and has enjoyed good health until some six or nine months ago, when his health began to fail, he became flatulent, dyspeptic, troubled with abdominal unrest, and began to lose flesh. Gradually attacks of colic came on, and became more frequent and severe as time went on. These attacks were accompanied by severe pain, abdominal distension, constipation, and perhaps by vomiting.

These attacks were, however, relieved by vigorous purgatives and enemas. The present attack commenced like the others, with a more or less gradual onset, and after some twelve hours or so reached a climax. When seen, the patient presents all the symptoms of acute intestinal obstruction, but in a less severe form than in strangulation; the pain is less terrible, the vomiting not so persistent and severe, and collapse not well marked. His bowels, on the other hand, are absolutely constipated, and enemas produce no result. Moreover, in marked contrast to nearly all cases of strangulation of the intestine, his abdomen is distended, and shews coils of intestine in vigorous peristalsis. This later sign clearly indicates that, however acute the present attack may be, progressive obstruction has existed for some months.

Opium.—The symptoms characteristic of the three forms of intestinal obstruction are clear and obvious, but they can all be made to disappear by the administration of opium in large doses without changing the gravity of the condition or preventing the progress of the case to a fatal termination. On a priori grounds opium should never be administered to an acute abdominal case until either the diagnosis has been established or the patient has finally refused operation. The result of the administration of a large dose of opium to a case of acute intestinal obstruction is surprising. The pain is relieved within an hour and the vomiting ceases, the patient grows warm and comfortable, the collapse disappears, and the pulse becomes once more full, regular, and strong.

It is often essential to know whether opium has been already administered to an acute abdominal case; for if so, allowance must be made in the consideration of every sign and symptom, and little weight attached to their absence in such a case. The signs which indicate the administration of opium are the pin-point pupil, the purple flushed cheek, and a dreamy tone of voice, easily recognised with a little practice.

4. The Diagnosis of Intestinal Obstruction.—The diagnosis of a case of intestinal obstruction may be divided into three stages. (i.) The General Diagnosis.—A careful review of the signs and symptoms of the case will shew whether there is mechanical obstruction of the intestine. (ii.) The Differential Diagnosis of the different Causes of Intestinal Obstruction.—When it has been proved that the bowel is obstructed, and by some organic and for the most part remediable cause within the abdominal cavity, the next step is to ascertain the nature of the cause. (iii.) The Differential Diagnosis from similar conditions.—The passage of food along the intestine may be arrested, however, by many conditions which are not classed under organic intestinal obstruction, and in the third stage of diagnosis these dynamic conditions must be excluded. Such are external hernia, peritonitis, pancreatitis, embolism and thrombosis of the mesenteric vessels, toxic conditions, and nervous derangements of the intestines.

(i.) **The General Diagnosis of Intestinal Obstruction.**—The diagnosis of intestinal obstruction is made from the presence of the symptoms considered in the last section. A careful review of the case

will not only shew that organic intestinal obstruction is present, but will also serve to place the case under its appropriate clinical heading of acute, chronic, or a chronic case which has terminated acutely. The common causes of each of these varieties may now be enumerated.

(A) *Acute intestinal obstruction* is usually due to intussusception, bands, volvulus, gall-stones, internal hernia, or apertures. (B) *Chronic intestinal obstruction* is usually due to cancerous stricture of the colon, faecal accumulation, chronic intussusception, stricture of the small intestine, pressure on the intestine from without. (C) *Cases of chronic intestinal obstruction which have terminated acutely.*—The causes of the chronic obstruction are those given above. The acute termination is due to the impaction of a foreign body, bending, kinking, or volvulus of the intestine above the stenosis, or to acute paralysis of the intestine. This classification is convenient, and for the most part trustworthy, but all the causes of acute obstruction given above may exceptionally produce subacute or even chronic symptoms, and they are then very liable to be wrongly diagnosed.

Up to this point the diagnosis of intestinal obstruction is comparatively easy, but the solution of the problem as to the exact cause responsible for the obstruction requires an investigation conducted with the most critical care, and it must be admitted that it is often impossible to come to a reliable conclusion.

(ii.) **The Differential Diagnosis of the different Varieties of Intestinal Obstruction.**—In order to discover which particular form of intestinal obstruction is present two methods may be adopted. The investigator may compare the descriptions of a complete list of causes with the case in point, and select that which most nearly tallies. This method of diagnosis is only possible for a medical man who from long experience is able to recognise diseases, as he does faces, at sight; others must be content to review carefully each point in the history, symptoms, and signs in turn, and thus determine to which particular cause it points. A review of the information so gained will frequently lead to an almost certain diagnosis.

A *history* of previous localised peritonitis will suggest that the obstruction may be due to a peritoneal or omental band or to adhesions, kinks, or bends. Under this heading come appendicitis, pelvic peritonitis, cholecystitis, and tuberculous peritonitis. External hernias which have been strangulated and then reduced are a prolific cause of intestinal obstruction by omental and peritoneal bands, whilst in other cases a hernia has been reduced *en masse* within the abdomen just before the onset of the symptoms of obstruction. A history of long-continued pain in the region of the liver, and perhaps of jaundice and colic, can be obtained in practically every case of gall-stone intestinal obstruction (*vide* p. 746). Definite symptoms of cancerous stricture of the colon are usually preceded by some six months of failing health, in a middle-aged patient, associated with abdominal unrest, dyspepsia, flatulence, irregularity of the bowels, and wasting. In other cases a definite history

may be obtained of dysenteric or tuberculous ulceration of the intestine which may indicate the probable cause of a stenosis. In very rare instances a history may be obtained of a separated intussusception or the passage of an enterolith.

Symptoms.—Onset and Duration.—As the case can readily be classed as acute or chronic from the character of the onset and the symptoms, the causes of acute and of chronic intestinal obstruction given on p. 725 must then be recalled. When the *pain* is sudden in onset and continuous without any period of relief the case is one of acute intestinal obstruction, and the cause is one of those detailed above. On the other hand, when the pain is colicky and intermits, it is due to recurrent waves of peristalsis, and the obstruction is chronic and partial. The same holds good with regard to the degree and rapidity of onset of the *collapse*. When collapse is well marked within twenty-four hours some form of strangulation is as a rule indicated. As regards the position of the obstruction, it may be stated generally that the more frequent, profuse, and urgent the *vomiting* the higher up in the alimentary canal is the obstruction, whilst when vomiting is infrequent or only occurs after food, it may be inferred that the obstruction is low down in the alimentary canal. The highest cause of obstruction is the passage of a large gall-stone from the gall-bladder directly into the duodenum, and the vomiting in these circumstances can only be described as tremendous, and it is often said to be faecal from the start; but as the stone passes down the intestine the vomiting becomes less severe. Bands, internal hernias, and apertures implicate the jejunum and ileum, and in these conditions the vomiting is frequent, severe, early becomes faecal, and is comparable to the vomiting seen in a strangulated external hernia. Intussusception is nearly always in the ileo-caecal region, and vomiting is not a special feature. The child usually vomits two or three times directly after the onset, but after that there are often long intervals, and the vomited matter only becomes faecal after several days. Malignant stricture is in the great majority of cases in the sigmoid flexure or the rectum, and accordingly the vomiting is late, infrequent, and only after food. Indeed, patients often say that since they took to a fluid diet they have not been sick at all. In volvulus of the sigmoid flexure, its most usual position, vomiting is of very subordinate importance, and thus contrasts with the acute character of the onset and of the other symptoms. In a case under my care the patient did not vomit at all during the whole course of his illness. When volvulus of the caecum is present vomiting is about as prominent as it is in intussusception.

Flatulence and rumbling of wind about the abdomen with difficulty in the passage of flatus by the bowel point to a partial obstruction, probably due to a stricture, a kink, or subacute strangulation beneath a band. The amount of distension and the size and situation of the distended coils of intestine will help to indicate the position of the obstruction.

When it is clear from the other signs and symptoms that a carcinoma

is present in the colon the situation of this carcinoma can be settled with a considerable degree of certainty by a careful inquiry. The patient will frequently describe how the wind invariably rumbles along a certain course in the abdomen and is arrested at a certain point where at the end of a wave of contraction a griping pain is felt. Sometimes he describes a mass which rises up at such times and is dispersed by rubbing with the hands. After such a spasm he says the wind disperses upwards and is not passed. When such rumbling and pain are confined to the right loin we may infer that the obstruction is at the hepatic flexure. When they pass up the right loin and across the upper part of the abdomen and the pain is in the left hypochondrium a carcinoma of the splenic flexure is usually present. When the wind rumbles down the left loin and the pain is felt in the hypogastric or left iliac region the obstruction is probably in the sigmoid flexure or upper part of the rectum. This latter group of symptoms is that more often encountered. *Tenesmus* indicates that the colon is implicated; it occurs in obstruction due to intussusception, volvulus, cancer of the colon, and pressure on the colon from without. The passage of *blood and slime* points to intussusception or to ulceration of the colon which, although more often malignant in cases of intestinal obstruction, may be innocent and due to dysentery or syphilis. The two common causes of blood and slime in cases of obstruction are cancer and intussusception, which are easily distinguished by the age of the patient, and the presence of a sausage-shaped tumour in 75 per cent of intussusceptions.

Signs.—Age.—When a baby, whose anus and rectum are apparently normally formed, suffers from its birth from either a total obstruction of the bowels or a very advanced degree of constipation, it may be inferred that a congenital malformation of the intestine is present. Such malformations take the form of breaches of continuity, septa, strictures, or in the case of congenital idiopathic dilatation of the colon it is probably a diminution or absence of the nervous reflex action of defecation. It is highly probable that intestinal obstruction in a baby during the first two years of life is due to intussusception, especially if it be a male child. Meckel's diverticulum causes trouble for the most part in childhood or in early adult life. Patients with cancer of the colon are as a rule over 40, but it must always be remembered that this form of carcinoma may occur at an earlier age than any other. Faecal accumulation is a disease of later life. The only form of acute intestinal obstruction which occurs almost invariably at an advanced age is that due to impaction of large gall-stones in the intestine. The average age of these patients, who are nearly always fat women, is about 60 years. (*Vide* p. 744.)

The amount of *distension* is usually an indication of the position of the obstruction in the alimentary canal, and of the chronicity of the condition. Thus, the greatest degree of distension is reached in congenital constipation persisting into adult life; one such patient, who was popularly known as the balloon man, attained to the gigantic girth of 86½ inches (Formad). Cases of acquired faecal accumulation also shew

very great distension. Cancerous strictures of the rectum and sigmoid flexure frequently lead to huge distension of the abdomen. It may be stated indeed that great distension in intestinal obstruction always points to some cause in the colon, and that it is seldom very great unless the cause be in the lower colon and of considerable duration. A partial exception to the above statement is volvulus of the sigmoid flexure, in which the onset of distension is very acute and the abdomen becomes exceedingly tense in one or two days; but in this case the distension is not the result of slow accumulation of faecal matter above the obstruction, but of the rapid formation of gas in the twisted intestine due to interference with its circulation. Cancer of the ileo-caecal valve, chronic intussusceptions, simple strictures and kinks of the lower ileum, and the subacute and chronic forms of incarceration under and over a band implicating the same part of the intestine may lead to very considerable distension of the abdomen, but in these cases the characteristic ladder arrangement of dilated small intestines is seen although the lower coils may reach so large a diameter as to be mistaken for large intestine. Acute strangulation by bands, internal hernias, or apertures usually occurs in the middle and upper parts of the ileum, and is seldom associated with distension until peritonitis has set in and has produced paralysis of the gut. Large gall-stones during their passage down the jejunum produce no appreciable distension. If they remain impacted in the lower ileum for several days the abdomen may become greatly distended, especially when peritonitis has supervened.

Tumours are sometimes felt in association with intestinal obstruction; in intussusception the characteristic sausage-shaped swelling may be felt under an anaesthetic in about three-quarters of the cases, and usually in the left loin. In obstruction due to pressure upon the intestine from without the tumour or swelling can often be felt on a careful abdominal examination, if the distension is not too great, or by the vagina or rectum when the tumour is wedged in the pelvis. Such swellings are the pregnant uterus, fibroids of the uterus, cysts and tumours of the ovary, sarcoma and cancer of the prostate, pelvic abscesses, retroperitoneal sarcoma, tumours, cysts, and abscesses of the kidney, and hydatids in various situations. Gall-stones causing intestinal obstruction have been felt in the region of the ileo-caecal valve or in the pelvic pouch in isolated instances. Cancers of the colon can rarely be palpated on account of the greatly distended pouch above them. Volvulus and acute dilatation of the caecum complicating a cancerous stricture of the colon can often be felt as a definite rounded swelling the size of a foetal head, and resonant on percussion.

Contracting coils of intestine, seen through the abdominal wall and felt to harden, indicate that there is partial obstruction of at least some days' duration. Coils of normal intestine are sometimes seen slowly contracting through a greatly emaciated abdominal wall, but differ from those indicating obstruction in the absence of dilatation and of hardening as they contract.

Contracting coils of large intestine are found in the region of the abdomen appropriate to them, and they are usually over three inches in diameter when they harden. They are most frequently seen in the case of a carcinoma of the upper part of the rectum or the lower part of the sigmoid flexure, and are situated in that case in the left iliac fossa or the hypogastric region. It should be remembered that waves of peristalsis pass in the transverse colon from right to left. A dilated stomach frequently occupies the same region of the abdomen, but in this case the waves pass from left to right. Visible contraction of the transverse colon is, however, rare. When it does occur it is often due to a cancerous stricture at the splenic flexure, and the waves do not then pass to the descending colon and the sigmoid flexure. The caecum may be seen and felt dilated and contracting in some of the subacute cases of dilatation of that viscus. The malignant stricture in such cases is frequently at the hepatic or splenic flexure of the colon, although it may be in the sigmoid flexure. In most cases, however, the dilatation is so acute and the distension so great that the organ is seldom able to contract. Faecal accumulation may give rise to a contracting colon, usually in the sigmoid flexure, which may become so greatly dilated as to occupy almost any part of the abdominal cavity, but perhaps the commonest position of the contracting coil is below and to the right of the umbilicus. The upper parts of the colon are seldom seen or felt to contract in faecal accumulation, although they may be greatly dilated.

Contracting coils of small intestine assume the well-known ladder arrangement, and are located in the central regions of the abdomen. The largest coils, which stand out prominently at the end of a wave of peristalsis, are as a rule on the right side of the abdomen below the umbilicus. These lowest coils in chronic cases may be dilated to a diameter of $2\frac{1}{2}$ to 3 inches, and it is often hard to believe that they are indeed small intestine. The cause of the obstruction is seldom lower than the ileo-caecal valve. In other words, the small intestine is seldom seen dilated and contracting as the result of obstruction in the large intestine, although this may occur in rare instances as shewn in a case figured by Nothnagel. The great capacity of the caecum and the tenuity of its wall appears usually to act as an efficient block between these two sections of the intestine. The causes of contracting coils of small intestine are chronic intussusception at the ileo-caecal valve, malignant stricture of the ileo-caecal valve and small intestine, simple strictures, adhesions, kinks, bends, and incarceration over or under a band, the latter causes being often the result of appendicitis.

A rectal examination will sometimes reveal the cause of intestinal obstruction, and should never be omitted. Cancer of the rectum may be felt or seen by means of a rectal speculum, and the sigmoidoscope (*vide* p. 858) reveals cancerous tumours which are far beyond the reach of the finger, but are suitable for resection. Foreign bodies, such as Murphy's button, enteroliths, faecal masses or large gall-stones, or the lower end of an intussusception may be felt in the rectum. Ballooning

of the rectum is often associated with malignant stricture low down in the colon, in the sigmoid flexure, or upper part of the rectum. It is, however, found in peritonitis, and many other conditions in which there is no true obstruction. I have seen it in acute colitis in a young man.

Enemas were formerly employed by Brinton and others to determine the position of strictures in the colon. The quantity of fluid the patient could retain was ascertained and the position of the stricture was estimated thereby; when more than a certain amount was retained the obstruction was supposed to be in the small intestine. Experience has shewn that this method is quite unreliable. Auscultation of the colon after the administration of an enema is but little less untrustworthy.

The passage of a soft bougie is another method which has been abandoned as useless and misleading.

When the other conditions, especially peritonitis, which produce *indicanuria*, have been excluded, excess of indican in the urine on the second day of an acute obstruction shews that the small intestine is implicated, and the absence of an increase of indican after three or four days points to a cause in the colon. It is of little value as a localising sign in chronic obstruction of the colon, for the amount is increased when the distension reaches up to the small intestine (*vide* p. 720).

(iii.) **The differential diagnosis of intestinal obstruction from similar conditions** is sometimes a very complex and difficult matter. Intestinal obstruction is mistaken for other conditions or conversely for the following reasons. (1) All violent stimulations of the abdominal sympathetic produce the same symptoms of sickening pain, vomiting, collapse, constipation, and frequently distension. It does not matter whether that stimulation is produced by the strangulation of a coil of intestine or by a renal or biliary calculus, an acute infection of the peritoneum, or the torsion of a testis or an ovarian tumour. (2) It might be supposed that in these latter cases as no organic obstruction exists the bowels would remain open, but it has been abundantly demonstrated by clinical and experimental evidence that all stimulations of the abdominal sympathetic powerfully inhibit the movements of the intestine (Bayliss and Starling), and cause spasmodic closure of the ileo-caecal valve (Elliott). Such spasmodic and paralytic forms of obstruction are sometimes most difficult to distinguish from those of organic origin. (3) Further, since the mechanism of intestinal peristalsis is most complicated and extensive, consisting of a complex neuro-muscular apparatus with an extensive vascular supply, it is not surprising that it should be liable to be thrown out of gear by the most diverse causes acting at diverse points. Thus, an embolus may block its arterial supply; thrombosis may invade its veins; some poison may act specifically upon its nerves, or these may become functionally deranged or be lacerated and stimulated by a mesenteric haemorrhage or inflammation. In whatever way the peristaltic mechan-

ism is inhibited, stasis follows with its symptoms of distension, vomiting, and constipation, and violent sympathetic pain and its reflex results frequently further complicate the diagnosis.

The diseases which have been mistaken for organic intestinal obstruction or conversely are innumerable, but the more important may be classified as follows :—

(*a*) An organic obstruction is actually present, but for purposes of treatment it is not called intestinal obstruction. In acute intestinal obstruction the presence of an *external hernia* must always be excluded by the routine examination of the inguinal, femoral, and umbilical rings, and by palpating the front of the abdomen to exclude a small ventral hernia passing through the linea alba, lineae transversae, or lineae semilunares, or buried in a fat abdominal wall. Scarpa's triangle should be deeply palpated for an obturator, and Petit's triangle for a lumbar hernia ; the gluteal region should be explored for a sciatic hernia. Strangulated hernias are almost always exceedingly tender, so that the patient indicates their position, except in the aged and in those profoundly collapsed. When the hernia is only incarcerated, however, there may be little local pain, but the severe pain from the coil of intestine may be referred to a point as high as the epigastrium in the eighth or ninth dorsal spinal areas. A patient under my care with a small incarcerated femoral hernia concealed in a fat groin, had, in ignorance of what was the matter, actually blistered his epigastrium to obtain relief ; his abdomen, which shewed visible coils of intestine, was opened and the hernia reduced. It is a serious matter to mistake *faecal accumulation* for obstruction due to some other organic cause, since laparotomy performed for faecal obstruction is nearly always followed by a fatal result. This mistake can nearly always be obviated by the routine practice of giving two turpentine or strong brine enemas in every case of obstruction before making a final diagnosis. The presence of compacted faeces in the rectum or putty-like tumours in the sigmoid flexure, transverse colon, or caecum, with a history of habitual constipation and of attacks of distension and vomiting relieved by purges and enemas, should make a diagnosis of faecal obstruction certain.

(*b*) The peritoneum has been violently stimulated by infection as in acute peritonitis due to the perforation of the appendix, stomach, or duodenum, or to tuberculous peritonitis. *Acute peritonitis* may closely resemble acute intestinal obstruction in its sudden onset with acute abdominal pain and collapse, and in severe vomiting and constipation. The vomiting, however, seldom becomes faecal, and an enema will shew that the constipation is partial. Peritonitis differs from obstruction in that the temperature is as a rule raised, the pulse rapid and wiry, and in the presence of the tenderness and rigidity of the abdomen. None of these signs is, however, absolute. The temperature in peritonitis may be normal or subnormal, the pulse between 80 and 90 and of good volume even in general purulent peritonitis, and even tenderness and rigidity may be absent or nearly so. As a matter of fact, however, uncom-

plicated cases of acute peritonitis are seldom diagnosed as intestinal obstruction or vice versa. Intestinal obstruction may complicate acute peritonitis from its very onset. I have pointed out that even in mild attacks of appendicitis the last coils of the ileum may be so inflamed, kinked, or twisted and fixed as to produce complete intestinal obstruction from the commencement of the attack. On the other hand many cases of intestinal obstruction are complicated by peritonitis. In strangulation of intestine from whatever cause peritonitis is usually present after the third or fourth day. Cancerous stricture of the colon frequently terminates acutely by perforation either just above the stricture or in the caecum. Such mixed cases of intestinal obstruction and peritonitis are exceedingly difficult to diagnose correctly.

Tuberculous peritonitis in its ordinary forms is not difficult to distinguish from obstruction, but in children there may be acute and transient forms which are usually mistaken for intussusception; possibly they are due to the rupture of a caseous tuberculous gland in the mesentery. The patients are delicate children from three to ten years of age, in whom, after a period of ill-health lasting about a week, there is a sudden onset of severe abdominal pain, constant vomiting, and constipation often resistent to purges and enemas. The tongue is moist and creamy, the temperature about 100° F., and the pulse good. The abdomen is tumid and slightly distended, not tender, and with the stethoscope the intestine can be heard to contract. Free fluid can often be detected in the peritoneal cavity and there may be palpable glands in the abdomen. A turpentine enema is usually effective, and the little patients rapidly convalesce (1). Tuberculous peritonitis may produce acute obstruction by the formation of peritoneal or omental bands, or by general matting and adhesions.

(*c*) The abdominal sympathetic system has received a violent sensory stimulus, as in *torsion of the testicle, ovary, or omentum, or in the passage of a biliary or renal calculus.* It has been pointed out that the symptoms of all violent stimulations of the abdominal sympathetic are, during the first few hours, almost identical whatever the cause may be. The history will, however, often assist in a diagnosis, and as the case progresses differential signs appear. In torsion of an undescended testis the organ is missing from the scrotum, and may be found at the internal ring or in the inguinal canal. In a twisted ovarian tumour there is often a history of a previous swelling in the abdomen, and the engorged tumour is nearly always palpable. Moreover, the symptoms of internal haemorrhage, such as blanching, restlessness, and a wiry pulse, are often present when the tumour is large or has ruptured. In the case of a twisted ovary, testis, or omentum, a turpentine enema will be followed by the passage of some flatus and faeces. In biliary colic there is often a history of previous attacks, and the distribution of the pain is more or less characteristic, and the pain may be followed by jaundice. The same remarks apply to renal colic in which alterations in the urine, especially haematuria and frequency, are seldom absent. This class of

case does not often cause any difficulty in diagnosis after the first twelve or twenty-four hours.

(*d*) The sympathetic nerves in the mesenteries have been violently stimulated as in *acute pancreatitis and haemorrhage into the mesentery.* The pancreas lies at the root of the mesentery and transverse mesocolon, and is in intimate relation with the coeliac plexus and semilunar ganglia. In many cases the destructive necrosis and haemorrhage spread between the layers of the mesenteries rather than to the peritoneal cavity. Many attacks of acute haemorrhagic pancreatitis are apyrexial. The irritation of the sympathetic ganglia and plexuses produces severe pain, collapse, vomiting, and a small and feeble pulse. The invasion of the roots of the mesenteries irritates the branches of the splanchnic nerves to the small intestine and colon, and these become distended and their movements inhibited so that almost total constipation results. In acute pancreatitis, however, the pulse is extraordinarily weak compared with the other symptoms; the temperature is often raised; a tender swelling may be felt deep in the epigastrium, and an enema usually produces a result. It may be said that when the peritoneum is not much invaded in acute pancreatitis it is more often than not mistaken for acute obstruction. Glands or hydatids in the mesentery may become acutely inflamed or haemorrhage may occur into that structure under exceptional conditions. My colleague, Mr. Roxburgh, performed laparotomy on a child with the symptoms of intestinal obstruction with melaena, and found haemorrhage into the mesentery. Next day the child developed a haemorrhagic small-pox rash.

(*e*) Some irritation of the gastro-intestinal mucous membrane is present as in arsenical and other forms of *poisoning*, and in some epidemics of *cholera*, *enteric fever*, and *colitis.* Acute strangulation has often been mistaken for poisoning, especially by arsenic, when the attendant circumstances were suggestive, and in some instances, as in three cases quoted by Leichtenstern, the true state of affairs has only been discovered at the autopsy. During epidemics many cases of very acute obstruction have been diagnosed as cholera, especially when characterised by anuria and death early in the disease. The mistake is still more easy when diarrhoea is present with obstruction, as is very rarely the case. A recent typhoid epidemic in the East End of London was characterised by an acute onset with profuse vomiting, severe colic, and constipation. Many cases were apyrexial, or nearly so at first. Several cases were diagnosed as acute appendicitis, and one at least was explored as possibly a case of intestinal obstruction. Typhoid spots appeared later in this case, and a positive agglutination reaction was obtained.

Simple colic due to the ingestion of indigestible articles of diet in adults or in children has often been mistaken for acute internal strangulation. The vomit, however, is never faecal, nor the constipation absolute, though enemas may be retained from the shortage of fluids in the body. Acute colitis in children may readily be mistaken

for intussusception from the association of vomiting with collapse and the passage of blood and mucus, especially when a tumour be felt in the abdomen. In one case of this kind, in which I opened the abdomen, the tumour turned out to be the right kidney covered by peritoneum on three sides. In other cases the contracted sigmoid flexure may be palpable but its diameter is less than that of an intussusception. In colitis the temperature is almost constantly raised from the start, the onset is not so sudden, and bile is found upon the napkin with the blood and slime. In acute intussusception, after the first motion or two, pure blood and mucus are passed and the temperature is as a rule not raised until the second or third day. Cases of chronic intussusception are almost always diagnosed as chronic colitis, at least for a time; faecal matter is mixed with mucus and blood in both cases. The presence of a sausage-shaped tumour in intussusception is the diagnostic sign, but I have known the decision delayed until the ileo-caecal valve presented at the anus.

(*f*) Some toxic cause has acted on the neuro-muscular mechanism of the intestine, as in lead poisoning and uraemia. To avoid mistaking *lead colic* for acute intestinal obstruction the gums should be examined for a blue line and inquiry made as to the trade of the patients. The subjects of lead colic are usually painters, white-lead workers, or have habitually handled old corroded lead, and many give a history of previous attacks in themselves or their fellow-workmen cured by castor oil and opium. An enema is usually successful. It must be remembered, however, that lead workers may suffer from intestinal obstruction, appendicitis, and peritonitis (*vide* Vol. II. Part I. p. 1046), and that their previous history is then very misleading.

Uraemia may closely simulate either acute or chronic obstruction. The points of similarity are profuse and urgent vomiting, well-marked constipation, pain in the abdomen and back, collapse, and not uncommonly in the chronic form of uraemia, distension. Careful examination of the patient and of the urine should lead to the recognition of uraemia. The mental condition is, moreover, usually more obscured in uraemia and suggests a toxic condition. If coma be present a mistake can hardly occur. I have seen acute dilatation of the upper colonic segment with profuse vomiting and total constipation in chronic uraemia. In another case a pregnant woman with acute nephritis and anuria was operated on for profuse vomiting, complete constipation, and severe abdominal pain. The gynaecologist and surgeon had overlooked the anuria.

(*g*) The cause of the derangement of the neuro-muscular apparatus of the intestine is organic or functional nervous disease as in *tabetic crises, hysteria, and other obscure nervous diseases.* The alimentary canal is divided into segments by sphincters: (1) the pyloric, (2) the ileo-colic, (3) a sphincter probably in the region of the splenic flexure, and (4) the sphincter ani. The innervation and functions of these segments have been so far differentiated that it is not surprising that their functional derangement should be more or less distinguishable. The toxic conditions

which simulate organic obstruction probably act through these neuro-muscular mechanisms. Acute uraemia acts chiefly on the gastric segment; lead colic mainly on the enteric segment, whilst acute dilatation of the upper or lower colonic segments is seen in chronic uraemia. The purely nervous conditions which simulate organic obstruction shew indications of a similar distribution. Tabes in gastric crises attacks the gastric segment, and also produces severe constipation often associated with loss of tone in the sphincter ani, which is probably a derangement of the lower colonic segment. I performed laparotomy for profuse vomiting, complete constipation, and severe attacks of abdominal pain in a syphilitic and alcoholic man who suffered from lightning pains in the legs and was more or less insane. The following parts of the alimentary canal were found distended; the stomach, lower half of the ileum, and the colon as far as the splenic flexure. The rest of the intestine was contracted, the transition from the dilated to the collapsed intestine being abrupt. No organic cause of obstruction was found, and purgatives and enemas were subsequently effective. Acute dilatation of the upper colonic segment in otherwise healthy people has been described, associated with vomiting and complete constipation, and has lead to colotomy in the transverse colon. The nervous derangements of the intestine here described are closely allied to acute dilatation of the stomach (*vide* p. 528).

(*h*) The intestine is extensively paralysed by *embolism or thrombosis of the mesenteric vessels.* The abdominal signs and symptoms of embolism of the superior mesenteric artery and acute thrombosis of the portal vein are very similar. In thrombosis, however, a considerable quantity of blood-stained fluid is found in the peritoneum and the spleen is frequently enlarged. The history and associated signs of the cause must mainly be relied on to distinguish them. In embolism mitral stenosis or infective endocarditis is probably present, but the abdomen is normal until the absolutely sudden onset of the attack. In thrombosis there are signs of some cause such as cirrhosis of the liver, cholelithiasis, or thrombosis of other veins, and the acute attack is preceded by obscure abdominal pains and transient attacks of diarrhoea and vomiting. In their final and acute attack both conditions resemble intestinal obstruction in their sudden onset with severe pain followed by vomiting which is violent and soon becomes brown in colour. Distension is present and the bowels are often completely constipated for the last day or two. On the other hand, the attack may commence with diarrhoea and often with melaena. Haematemesis is common and there is usually a considerable quantity of free fluid in the abdomen. In the three cases of thrombosis and one of embolism that I have seen, organic intestinal obstruction was suggested by the symptoms, and in one it was actually diagnosed. Laparotomy was performed in two cases and all the patients died.

(*i*) The intestine is not obstructed, but vomiting and constipation are present, and a contracting viscus or a tumour is found in the abdomen as in pyloric obstruction, cancer of the greater curvature of the stomach, or the contracted form of carcinoma of the stomach. These three forms of

carcinoma of the stomach may simulate chronic obstruction of the transverse colon. Pyloric obstruction resembles chronic intestinal obstruction in the presence of obstinate constipation. The bowels are often closed for seven days at a time and then small hard scybala are passed. Purgatives fail to act, for they are retained in the stomach and provoke further vomiting. Vomiting is present at irregular intervals and frequently occurs after food, but its great quantity and fermenting character should direct attention to the dilated stomach. In pyloric obstruction an elongated organ in active peristalsis may be found at almost any level in the abdomen and may be mistaken for the transverse colon, but in gastric peristalsis the waves pass from left to right. By giving a teaspoonful of sodium bicarbonate followed by the same quantity of tartaric acid the outline of the stomach can be made visible.

Cancer of the greater curvature of the stomach, especially in a case of gastroptosis, may be mistaken for cancer of the colon. If the growth does not interfere with the orifices or movements of the stomach, gastric signs and symptoms may be almost absent. Moreover, carcinoma in this situation tends to spread along the great omentum to the transverse colon and may irritate or actually obstruct that organ. In a case under my care, the passage of mucus, blood, and scybala associated with a mobile tumour in the left iliac fossa and absence of vomiting lead to a diagnosis of carcinoma of the sigmoid flexure. A laparotomy shewed that the tumour was carcinoma of the greater curvature of a greatly displaced stomach, and that the transverse colon was surrounded by the growth. The form of carcinoma known as india-rubber bottle stomach may irritate or obstruct the colon in a similar way, but the contracted stomach is concealed beneath the liver and ribs and cannot be examined either by palpation or percussion after distension with gas. Moreover, the gastric condition produces persistent vomiting of small quantities. Such cases are seldom diagnosed correctly.

5. Terminations, Duration, and Prognosis.—**Terminations.**—Death is certain to follow in practically every case of organic intestinal obstruction which is not submitted to operation, with the exception of those due to faecal accumulation. It may occur early in the attack and is then due to collapse. In acute strangulation it is most often due to peritonitis the result of a crude perforation or of the passage of micro-organisms through the intestinal walls. Many patients succumb to toxaemia or to septicaemia, and where faecal vomiting has occurred not a few die of inhalation bronchopneumonia. Very rarely death has been brought about by phlegmonous processes and suppuration in the liver and lungs.

Duration.—A case of acute intestinal obstruction may terminate within a few hours or last as long as twenty-one days. The average duration of 350 acute cases collected by Leichtenstern was six days. Chronic obstruction may drag on for months or even years, and in faecal accumulation absolute constipation has persisted for as long a period as eight or nine months (Strong).

Prognosis.—*Spontaneous cure* may occur in any form of intestinal obstruction, but this is so exceedingly rare that it cannot be anticipated. It is more often seen in faecal impaction than in other forms of obstruction, and it certainly occurs in cases of compression of the intestine by tumours from without. Large gall-stones and foreign bodies are sometimes passed even after days of obstruction; occasionally in invagination the intussusceptum will slough away safely; and in both volvulus and internal hernia spontaneous recoveries have taken place.

Recurrence is a very unusual occurrence. It is more common in cases of volvulus and intussusception. The opinion that intussusceptions frequently recur is derived from the time when this condition was treated by forcible inflation. The intussusception was then only partially reduced. In recent years the majority of cases have been treated by laparotomy and direct reduction and recurrence is almost unknown; thus one case only has recurred at the London Hospital in recent times. The recurrence of symptoms may be due to an entirely new cause of obstruction, as in a case in which I performed colotomy for carcinoma of the colon, and some days later acute obstruction supervened due to strangulation of a coil of ileum by an omental band.

Mortality of Intestinal Obstruction after Operation.—More than half the cases of intestinal obstruction submitted to operation die. Of 669 cases of all forms of intestinal obstruction treated in the surgical wards of the London Hospital from 1893 to 1905, 366 died and 303 recovered, a mortality of 54·7 per cent. I am indebted to Dr. Holzmann for the following table shewing the mortality of a series of 100 consecutive cases of acute intestinal obstruction treated from 1900 to 1902 inclusive at the London Hospital:—

	Cases.	Died.	Recovered.	Mortality.
				Per Cent.
Strangulation by bands or through orifices	34	16	18	47
Intussusception	52	28	24	53·8
Volvulus	8	7	1	87·5
Impacted gall-stones	6	4	2	66·6
	100	55	45	55

These figures are derived from hospital records and therefore are nearer the truth than statistics of published cases, which are as a rule not consecutive. It should be emphasised, however, that of the fifty-five fatal cases in the above series no fewer than twenty-nine were admitted to the hospital collapsed after several days' obstruction; twenty-six of these died of shock soon after the operation, and three were not operated upon as they were moribund. If it were fair to assume that all these cases would have recovered if operated on earlier, the mortality of this series would fall to a little over 25 per cent. It may be stated, therefore, that when recognised early and operated on within two or three days of the onset,

the mortality of acute intestinal obstruction is about 30 per cent, or 1 case in 3, and that the mortality of chronic intestinal obstruction in similar circumstances is not higher than 15 per cent.

The following points bear on the prognosis of intestinal obstruction. (1) The age of the patient and his general vitality; (2) the duration of the illness; (3) the presence of collapse, toxaemia, or septicaemia; (4) the presence of peritonitis; (5) the presence of distension; (6) the cause of the obstruction, if this can be ascertained.

Age.—In babies and the aged the prognosis is bad. This is well shewn by the following series of 100 acute cases analysed by Dr. Holzmann:—

Age.	Cases.	Recovery.	Death.	Mortality.
				Per Cent.
Under 1 year old . . .	38	15	23	60·5
Over 50 years old . . .	14	5	9	64·3
Over 1 year and under 50 years	48	25	23	47·9
	100	45	55	55

Age, no doubt, accounts for the high mortality in intussusception (53·8 per cent), and in gall-stone intestinal obstruction (66·6 per cent).

Distension in acute intestinal obstruction of the small intestine is a most serious sign. It indicates not only that the operation will be difficult, but the probable existence of peritonitis, and the presence of a large quantity of toxic matter which must be evacuated before the patient is free from the danger of toxaemia. I have never seen a case of intussusception with distension in a baby recover. In chronic obstruction distension is not of such serious import.

The cause of the obstruction has an important bearing on the prognosis. The prognosis is probably worst in volvulus, while impacted gall-stones and intussusception are very serious forms of intestinal obstruction. Strangulation by Meckel's diverticulum is also very fatal, for gangrene of the intestine and peritonitis appear early in such cases.

In short, if the patient be a young adult with healthy viscera and has not been ill more than two or three days the prognosis after operation is good. On the other hand, if the patient be old and feeble, with a fatty heart, granular kidneys, emphysema, and the illness has lasted several days, the prospect is wellnigh hopeless.

6. The General Treatment of Intestinal Obstruction.—Excluding faecal impaction the treatment of organic intestinal obstruction is purely surgical, and will therefore be referred to here in the briefest manner only. The objects the surgeon has in view are to open the abdomen, determine the cause of the obstruction, and relieve it with the least possible loss of time. The median *incision* below the umbilicus is usually chosen, because through it practically all parts of the abdomen can be reached. To locate the cause of the obstruction the following method is usually adopted. The caecum is first examined; its distension indicates

that the cause is in the colon. The sigmoid flexure is next drawn out; distension shews that the obstruction is in the upper part of the rectum or lower part of the sigmoid flexure. Should the sigmoid flexure be collapsed the transverse colon is drawn into the upper angle of the wound; if this be distended the obstruction will be found at the splenic flexure or in the descending colon. If the transverse colon be empty whilst the caecum is distended, the cause of obstruction will probably be found at the hepatic flexure. When the caecum is found to be collapsed the obstruction is in the small intestine. The surgeon then as a rule draws out the collapsed coil of ileum entering the caecum, and traces it backwards to the obstruction. On the other hand, he may prefer to draw out a distended coil of small intestine, and having determined which is its lower end, follow it down to the cause of the obstruction.

The special treatment adapted to each cause of obstruction cannot be discussed here. The general principle which guides the surgeon when he has located and identified the cause is to relieve the obstruction at once, and to remove the cause if this can be done in a few minutes. Thus, a band may be divided, a gall-stone evacuated, or an intussusception or volvulus unravelled. If, however, the removal of the cause would take some considerable time as in the case of a carcinoma of the colon, the point of obstruction must be short-circuited by lateral anastomosis or the gut opened and drained above it. The cause may then be dealt with at a second operation should the patient recover from his intestinal obstruction. It would be wrong to leave the subject of the treatment of intestinal obstruction without insisting that opium should not be given to an acute abdominal case until a definite diagnosis has been made or the patient has finally refused operation.

Part II.—Special Forms of Intestinal Obstruction

Synopsis:—*A. Causes within the Lumen of the Intestine—Internal Obturation*—(i.) True Foreign Bodies; (ii.) Enteroliths; (iii.) Faecal Masses; (iv.) Gall-stones. *B. Causes in the Wall of the Gut—Internal Stricture*—(i.) Carcinomatous Stricture; (ii.) Cicatricial Stricture; (iii.) Congenital Stricture. *C. Causes outside the Intestine*—(i.) Anomalous forms of Adhesions; (ii.) Compression; (iii.) Herniaform intestinal obstructions due to (*a*) Bands, (*b*) Apertures, (*c*) Internal Hernias. *D. Intussusception. E. Volvulus of the Intestine*—Volvulus of the Omentum.

A. Internal Obturation of the Intestine.—Cases of this class of intestinal obstruction may be subdivided as follows:—Those due to (i.) true foreign bodies; (ii.) enteroliths: (iii.) faecal masses; (iv.) gall-stones.

I. **Intestinal Obstruction due to true Foreign Bodies.**—This is an extremely rare form of obstruction; thus, there was only one case among 669 consecutive cases of intestinal obstruction at the London Hospital in

the thirteen years 1893-1905 ; but in four further cases foreign bodies were arrested above strictures or bands.

Etiology.—The circumstances in which foreign bodies gain an entrance into the intestine are various, interesting, and bizarre. Children may swallow coins, pins, needles, brooches, pieces of coal, comforters, corks, beads, and even mice (Roy, G. G.). Accidents, especially during sleep or anaesthesia, account for the ingestion of many tooth-plates, and false eyes placed in a glass of water have been swallowed. The bones of fish, fowls, rabbits, sheep, and the stones of plums, cherries, prunes, dates, and apricots are frequently swallowed with food. Burglars, thieves, and coiners have swallowed coins, watches, rings, jewels, and precious stones such as diamonds. Jugglers, from this point of view, may be divided into two classes : those who accidentally let slip large objects which they have introduced into their oesophagus, such as table-knives and forks, swords and sticks, and those who for small sums will really swallow pence, pipes, watch-chains, studs, pocket-knives, corks, string, straps, and other strange small objects. To the former class belongs the case of William Dempster who swallowed a table-knife at Newcastle by mistake (23). To the latter that of the "Human Ostrich," who swallowed coins, pipes, string, and used to eat bottles and plates in public-houses at the East End (Eve, F. S.).

Hysterical girls have been known to swallow keys, safety-pins, nails, needles, hair, finger-nails, and skeins of wool and cotton. Hair is quite indigestible, and is apt to collect into hair-balls or bezoars. Some hysterical women continually suck their hair, others pull it out and swallow it, others again cut the ends off and eat them. Several such hair-balls of great size are in the Royal College of Surgeons' Museum. Two masses of bent pins, weighing a pound and nine ounces, in the stomach and duodenum of a woman may be seen in the same collection. Several pins ready bent to be swallowed were found in and around the patient's death-bed. Lunatics swallow most unsuitable objects, sometimes in order to commit suicide, at other times to appease unnatural appetites. Thus, a patient with a tumour of the prostate and base of the bladder suddenly became acutely melancholic, and in the course of one night he devoured nearly the whole of one sheet, and died in the morning.

Certain trades lead to the repeated swallowing of small indigestible objects which become matted into balls or masses. Dressmakers swallow pins, needles, and thread ; carpenters nails, and in the intestine of a boy who plucked fowls, and was in the habit of licking his fingers, a ball of down the size of a hen's egg was found (Specimen K. 7, Calculi, Roy. Coll. of Surgeons' Museum). Since their introduction, several Murphy's buttons have become impacted in the ileo-caecal valve and lower ileum, an accident foretold by Chaput.

Pathology and Symptoms.—The most useful classification of foreign bodies is into : (1) sharp and irregular foreign bodies such as tooth-plates and meat bones, which are liable to become arrested and to set up enteritis. In other cases they perforate the intestine and produce either

a local abscess or general peritonitis. (2) Smooth, ovoid, or round foreign bodies, such as fruit-stones, beads, and bullets, which pass safely along the alimentary canal unless arrested by a stricture or some other cause of stenosis, when they may readily produce acute obstruction. (3) Small indigestible objects which are prone to become matted into balls or masses.

The rate of progress of foreign bodies along the alimentary canal varies very greatly. (i.) The great majority pass uninterruptedly in from two to twenty days, and as a rule produce no symptoms in their passage. In some instances a little colic with mild nausea and vomiting has been present. (ii.) Some foreign bodies, for the most part irregular in shape, are retained for a longer or shorter period, and then escape by the natural channels. They are arrested near the end of the ileum, the ileo-caecal valve, or the caecum so that symptoms are referred to the right iliac fossa. They are usually retained for a period measured in months, but it may be as long as ten years. The symptoms produced by their arrest are those of ulcerative enteritis, localised or general peritonitis, but only very rarely those of obstruction. Even when the foreign body has been passed the patient may still die of ulceration or stenosis. (iii.) Foreign bodies may become permanently arrested in the normal intestine in the situations already indicated, but they often become arrested in other situations as the result of narrowing produced by stricture, adhesions, or the pressure of a tumour from without. In other instances foreign bodies are permanently arrested in hernias, diverticula, pouches, or loops. The results of the permanent arrest of a foreign body in the intestine are obstruction, enteritis, and perforation. Obstruction is the least common, and is nearly always partial and attended by colicky pain and vomiting, seldom by constipation or by abdominal distension, unless peritonitis coexist. A very large and round foreign body may produce acute intestinal obstruction as in the case of a man who swallowed an egg-cup. Ulcerative enteritis nearly always occurs, producing pyrexia, local tenderness over the impacted tumour, frequent fluid stools containing pus and blood, and rapid wasting. Intestinal perforation due to arrested foreign bodies scarcely concerns us here; some, such as needles, may perforate and wander to distant parts of the body without producing symptoms; some set up acute peritonitis; others pass into an abscess cavity outside the intestine and then pass through the skin or enter a hollow viscus such as the bladder, ureter, pelvis of the kidney, the vagina, or vena cava. Some foreign bodies become embedded in dense fibrous tissue, and may then simulate malignant growths.

The prognosis in swallowed foreign bodies which have reached the intestine is exceedingly good. Nearly all are safely evacuated. Death when it does occur is due to the following causes arranged in the order of their frequency: — (1) Marasmus and wasting due to ulcerative enteritis; (2) peritonitis and abscess-formation; (3) least often intestinal obstruction.

The treatment should be persistently expectant, for a foreign body, able to travel through the oesophagus and pylorus, can almost certainly

pass the ileo-caecal valve and anus. It is customary to administer coarse food such as porridge and vegetables, and to avoid fluids and purgatives whilst the body is on its way. There is no doubt that the advances of abdominal surgery and the introduction of *x*-rays have been responsible for many operations, which, unless the foreign body has become arrested or is producing symptoms, are unnecessary. A skiagram to be useful should be taken immediately before operation, otherwise it will often be found that the foreign body has moved on and may have reached the rectal pouch. Abscesses should be opened and drained. When large foreign bodies are present in a sinus their exit should not be hurried unduly.

II. **Intestinal Obstruction due to Concretions, Calculi, and Enteroliths.**—This condition is exceedingly rare. One case has occurred in 669 cases of intestinal obstruction at the London Hospital in thirteen years.

Etiology and Pathology.—The conditions which govern the formation of intestinal concretions are as follows:—(i.) A nucleus which is nearly always of an organic nature; such nuclei are fruit-stones, masses of vegetable or animal hairs, or gall-stones. (ii.) A cause of arrest is a necessary condition. The vermiform appendix or a saccule in the colon or caecum frequently arrests such a nucleus. The lower end of the ileum is another favourite site for arrest, probably in consequence of its dependent position in the pelvis or as a result of adhesions from appendicitis or salpingitis. (iii.) Adhesive particles must be supplied continuously in the food. These particles are sometimes thread-like structures, such as hairs, wool, and vegetable fibres of cotton, hemp, and the setae of the oat seed. Nearly all these threads have a spiral structure which enables them to interlock. Other particles adhere by a process of crystallisation; such are magnesium and iron salts, salol, and other drugs. (iv.) Chronic enteritis must be started and maintained around the growing concretion. The chronic irritation provides an organic element and a colloid menstruum in which crystallisation readily takes place. The bacterial decomposition of proteins produces ammonium phosphates and carbonates, and in the presence of calcium or magnesium salts triple phosphates are readily produced.

Intestinal concretions, calculi, or enteroliths may be classified as follows:—(1) Concretions formed from some habitual element of the food which progressively accumulates. Oat, fat, starch, and meat-balls are examples. (2) Concretions formed from amorphous faecal matter. (3) Those composed of minute foreign bodies frequently swallowed, such as threads, string, hairs, and wool. (4) Concretions may be produced by a prolonged course of certain drugs, such as magnesium and iron salts, gum benzoin, liquorice root, salol, and amalgams administered for worms. (5) Calculi which are hard like stones, and are composed of concentric layers of phosphates and carbonates of calcium, magnesium, and ammonium with an organic basis. These are almost entirely the result of the chronic inflammatory processes set up by the presence of a nucleus

in an intestinal diverticulum or pouch. (6) Balls of mucus have been described as a cause of intestinal obstruction. These were probably cases of mucous colitis. (7) Balls composed of the *Ascaris lumbricoides* have also been described, but it is very doubtful if they ever occlude the intestine.

The *symptoms* of a progressively enlarging concretion differ somewhat according as it is in the ileum or in the colon. When the concretion is in the ileum the history is one of successive and progressively severer abdominal attacks. In the earlier attacks the symptoms are rather those of enteritis, colic, pyrexia, diarrhoea, and a variable degree of vomiting. As the condition progresses obstructive symptoms such as distension, visible and contracting coils of intestine, severe vomiting, and constipation during the attack begin to be more prominent. A tender swelling may also be found in the right iliac region or on a rectal or vaginal examination. As the case goes on and ulceration occurs around the stone, pus and blood appear in the stools. The final attack of complete obstruction is probably due to a displacement of the concretion from a pouch into the lumen of the gut, or to spasm of the intestine due to an acute attack of enteritis.

When the concretion is in the colon the symptoms are those of attacks of ulcerative colitis, severe colic, tenesmus, and the frequent passage of watery stools containing pus and blood. The patient wastes progressively, and a tender swelling is more often palpable than when the concretion is in the small intestine. Symptoms of obstruction are rare and only occur very late.

Treatment.—This condition is seldom diagnosed before an operation or an autopsy. Concretions in the rectal pouch may be broken up and evacuated, and for this purpose it may be necessary to dilate the sphincter forcibly. When the concretion is higher up treatment is purely surgical; if possible, the concretion should be displaced upward from the region of ulceration; the intestine is then incised and the mass removed.

III. **Intestinal Obstruction due to Faecal Masses.**—Since chronic constipation and idiopathic dilatation of the colon are described elsewhere, it is only necessary to point out here that when obstruction is due to faecal masses the primary cause is an atonic condition of the intestinal muscle which permits of the faecal accumulation, and that the faecal masses and the atonic bowel both play a part in the final stage of intestinal obstruction. Of 669 cases of all varieties of intestinal obstruction admitted to the London Hospital in thirteen years, 69 were due to faecal accumulation. The proportion of the sexes was about equal, viz. 32 men and 37 women. Two cases died, a mortality of a little less than 3 per cent.

IV. **Intestinal obstruction due to gall-stones** is said to occur when a large gall-stone becomes impacted in the alimentary canal. Mr. Mayo Robson gives three other associations of intestinal obstruction with gall-stones:—(1) volvulus of the intestine due to gall-stone colic or to the passage of a calculus along the intestine; (2) stenosis of the intestine

due to adhesions around the gall-bladder or the healing of gall-stone fistulae; (3) obstruction dependent on local peritonitis, in the neighbourhood of the gall-bladder, leading to paralysis of the intestine.

Incidence.—This is a rare form of intestinal obstruction. At the Leeds Infirmary there was one case in ten years, and at St. George's Hospital but one in fifteen years. At the London Hospital 15 cases have occurred out of a total of 669 consecutive cases of all varieties of intestinal obstruction in thirteen years, a proportion of 1 in 44·6. Authorities who have derived their series from the literature consider the condition much more common: Fitz, 1 in 13; Gibson, 1 in 17; Leichtenstern, 1 in 28. The proportion of women to men in this variety of obstruction is 5 to 1, and is the same as the ratio of the sexes in cases of gall-stones in the gall-bladder. In the series of 15 cases at the London Hospital the proportion was only 4 women to 1 man. The average age of this same series was 59·3 years. Sir F. Treves gives the average age as 57, Mr. Eve as 64, and Dr. Rolleston as 62·7.

Morbid Anatomy and Pathology.—The Method of Entrance of the Calculus into the Intestine.—Courvoisier gives the following result of 36 autopsies:—The stone had passed through a fistula between the gall-bladder and the duodenum in twenty-five cases, through one between the gall-bladder and the ileum in one case, through one between the gall-bladder and the colon in one case, through one between the gall-bladder and the duodenum and the colon in two cases, and along the common bile-duct in seven cases. He dismisses three of these last seven cases as dubious, but unhesitatingly accepts the four remaining cases, in which the gall-bladder was not adherent to the intestine. In one case the impression of the two obstructing calculi was found in the common bile-duct; in two cases the common bile-duct was as large as the gall-bladder, and in one of these cases the two cavities were continuous. In two cases it was clear that the calculi had formed in the bile-duct. The calculus almost invariably passes through a fistulous opening from the posterior surface of the neck, body, or fundus of the gall-bladder into the first part of the duodenum, that is to say, the part of the duodenum which is so often bile-stained at autopsies. The encysted stone first leads to adhesive peritonitis around the gall-bladder, which becomes fixed to the contiguous duodenum; the wall between the cavities is then attenuated by inflammation until a communication results. The calculus projects into the duodenum for a longer or shorter time before it escapes; on the projecting end chyme and phosphates are deposited until in some cases the top of the calculus becomes white, mulberry-shaped, and like a nipple (30). The final displacement of the stone has in several cases followed the administration of a purge, and in one case was apparently due to the passage into the common bile-duct of a smaller calculus, which diverted the flow of bile through the gall-bladder, and so loosened and evacuated the large calculus into the duodenum. In a smaller proportion of cases the fistula forms between the gall-bladder and the transverse colon. The calculus is then voided spontaneously,

and in the majority of cases without symptoms of intestinal obstruction. It is stated that such stones have become impacted in the sigmoid, and not uncommonly they remain for some time at the anus, and require digital extraction. When a stricture of the colon exists even a small gall-stone which has passed along the common bile-duct may occlude the narrow lumen left. In very rare instances the fistulous opening has connected the gall-bladder with a coil of the jejunum.

It is stated that a gall-stone may pass into the bowel and remain there more or less latent for years before producing acute intestinal obstruction. Presumably the calculus is encysted in a pouch, and during its residence in the bowel it is washed nearly free from bile-pigment and covered with phosphatic accretions. Such stones are supposed to reveal their presence by attacks of colic, vomiting, constipation, and sometimes distension. The recorded cases are few and the evidence doubtful.

The Spontaneous Passage of large Gall-stones per Anum.—It is stated that 50 per cent of cases of gall-stone intestinal obstruction when not operated on are relieved by the spontaneous passage of the calculus, sometimes quite suddenly (Rolleston). The patient frequently sinks nevertheless. The spontaneous passage of a gall-stone of an inch or more in diameter is not always preceded by symptoms of intestinal obstruction; in such instances the gall-stone has passed into the transverse colon and not into the duodenum. The proportion of such cases is stated by Sir F. Treves to be one-third, but Courvoisier considers it much rarer. The spontaneous passage of a gall-stone may be greatly assisted by the administration of drugs, such as opium, belladonna, and of general anaesthetics, which relax the spasm of the intestine beneath and around the gall-stone.

The Site of Impaction.—The point at which the calculus becomes impacted is usually the lower part of the ileum. The small intestine diminishes in diameter from the duodenum to the caecum, the ileo-caecal valve admitting a round object of about an inch or less in diameter. Since gall-stones are rare in proportion to their size the majority of impacted gall-stones are found about the ileo-caecal valve and the lower part of the ileum, a few in the jejunum, and only in rare cases in the duodenum. Courvoisier classified 53 cases as follows:—In the sigmoid flexure, 2·4 per cent; at the ileo-caecal valve, 10 per cent; in the ileum, 65·4 per cent; and in the duodenum, 21·4 per cent.

The Pathological Basis of the Symptoms.—The first symptoms, severe pain and profuse vomiting, are due to an intense irritation of the duodenum by the entrance of the calculus. The vomiting is more profuse than in any other form of intestinal obstruction, and aptly illustrates the general rule that the higher up in the intestine the obstruction occurs the more profuse the vomiting. The dark colour of the early vomited matter is probably due to haemorrhage from the edges of the fistula, and from the raw interior of the gall-bladder increased by the venous engorgement associated with the vomiting. In gall-stone intestinal obstruction there is no strangulation of the intestine. This is a pure obstruction of the

lumen of the small intestine without interference with its blood-supply and without paralysis of the nerves or muscles of the intestinal wall. Even this obstruction of the lumen is incomplete for the first few days whilst the calculus is passing along the upper and wider portions of the small intestine, and during this period the constipation is partial and the pain colicky and intermittent. When impaction of the calculus occurs the obstruction becomes complete, the constipation absolute, and the pain continuous.

The Manner in which Obstruction is produced.—Spasm of the intestine beneath and around the calculus is the chief cause of obstruction. At nearly all operations on such cases the stone is found resting on a septum formed by the contracted intestine beneath it, whilst the intestine above is widely dilated. On the other hand, at many autopsies on cases that have not been operated on the calculus has been found loose in the relaxed intestine, and it has been hard to understand how it can have produced obstruction at all. Morphine, belladonna, and general anaesthetics act by relaxing this spasm. Inflammatory swelling of the mucous membrane appears after a day or two and helps to make the obstruction complete. The small intestine, unlike the sigmoid, colon, and rectum, is not adapted for propelling hard rough masses; its interior is covered with the folds of the valvulae conniventes specially fitted to retard the progress of fluid contents. The surface of the calculus is also of importance; when it is rough and angular a smaller calculus will become impacted; when it is smooth and rounded a larger one will pass spontaneously. If a gall-stone remain for any length of time in the intestines its bulk will be increased by phosphatic and faecal accretions.

The Previous History of Biliary Trouble.—A gall-stone must be an inch or more in diameter in order to obstruct the intestine. Gall-stones of this size very rarely pass along the biliary passages, but as a result of a process of adhesion and ulceration work their way directly from the gall-bladder into the first part of the duodenum. This process gives rise to long-continued dull pain, with exacerbations, in the right hypochondrium and epigastrium. It is not very uncommon, moreover, for smaller stones to be associated with one or more larger ones, and these small calculi may enter the ducts and produce both colic and jaundice. In 9 cases at the London Hospital, in which the question of previous biliary trouble was specially investigated, 2 had had colic and jaundice, 2 spasms with vomiting, 4 dull dyspeptic pains, and 1 no symptom at all.

The Clinical Picture.—The patient is usually a fat woman about sixty years of age suffering from an acute obstruction of the small intestine without distension and of sudden onset. The pain during the first day or two is intermittent and colicky, shewing that the obstruction is partial, and that the intestine is not strangled. It is located in the epigastrium at first, but subsequently shifts to the umbilicus when the stone passes from the duodenum to the jejunum. The most prominent symptom is vomiting, which is severe, continuous, profuse, and is black or brown with blood or bile at the commencement of the attack. A gallon or more

may be ejected in a couple of days, a quantity greatly in excess of the fluids swallowed. In many cases the vomiting can be divided into three periods (Barnard). In the first it is sudden in onset, severe, continuous, and profuse in character and dark in colour. In the second, when the stone has moved down the intestine, and usually on the third day, the vomiting abates and becomes green. As the bowels are frequently moved on this day the patient believes she is about to recover. In the third stage the stone has become impacted, and the vomiting recurs, and is brown and stercoraceous. It is in this stage that the surgeon is applied to for relief.

The condition of the bowels varies; whilst the stone is moving faeces and flatus are able to travel past the calculus, and in most cases therefore the bowels act in response to purgatives and enemas during the first three days of the attack. About the fourth or fifth day the stone becomes impacted, and constipation becomes absolute. Collapse is seldom well marked until the fourth day of the disease. Abdominal examination reveals little save obscure tenderness around the gall-bladder. The slight distension cannot be appreciated in such fat subjects. The calculus is scarcely ever felt. It has, however, been palpated in the right iliac fossa and in Douglas' pouch, which is no doubt the usual position for a calculus which is dragged there by its own weight.

Diagnosis.—It is possible to diagnose gall-stone intestinal obstruction by the unusual grouping of symptoms it presents. An elderly fat woman, with acute but partial obstruction of the small intestine, violent onset with vomiting, and only partial constipation, migration of the pain from the epigastrium to the umbilical region, remission of the vomiting on the third day, and a previous history of biliary trouble. In the presence of all these symptoms the diagnosis may be made with confidence.

Duration, Prognosis, and Mortality.—When treated by narcotics and anaesthetics 50 per cent of cases are relieved spontaneously even as late as the twenty-eighth day (Sands). Many of these cases nevertheless succumb subsequently. In most cases not so relieved death occurs from the fifth to the tenth day. The mortality after operation is very high. At the London hospital 13 of 15 cases were operated on, and of these 8 died, a mortality of 61·6 per cent; I have operated on 7 cases with 4 recoveries. This high mortality is to be attributed to the great age of these patients and to their fat and degenerate organs, and to the late date at which they are submitted to operation owing to the remission of symptoms on the third day. The average age of the fatal cases at the London Hospital was 63, of those that lived but 53. Among the fatal cases the duration of obstruction was just under eight days on the average, and among the recoveries four days. No case in which obstruction had existed for five days or more recovered, and the only aged woman (73) who lived was thin. Tillmann states that recent results are much more favourable, and he quotes Körte as having had four recoveries in five consecutive cases. Operation on the second or third day would probably give very good results.

Treatment.—Every case in which acute intestinal obstruction is diagnosed should be operated on at once, and when it is thought to be due to a gall-stone early operation is all the more imperative for the reasons already stated. The gall-stone should be removed by a longitudinal incision through the intestinal wall, and this incision immediately closed by sutures. If operation be declined or be impracticable morphia and belladonna may be prescribed in large doses with a moderate chance of success. General anaesthetics, massage, and enemas have appeared to be useful in certain cases.

B. Internal Stricture of the Intestine.—*Definition.*—An internal stricture is a narrowing of the lumen of the bowel produced by changes in the wall of the intestine.

The lumen may be diminished in three ways: (1) It may be blocked by new growth projecting from the wall as in cancerous stricture. (2) The wall itself may be contracted, usually as the result of healing ulcers and cicatrisation after gangrene and injury. (3) The wall may be so thickened by cancerous, tuberculous, or gummatous tissue that the lumen of the bowel is greatly narrowed. It is customary to exclude strictures of the rectum from a description of intestinal obstruction. Internal strictures of the intestine are divided into (1) carcinomatous, (2) cicatricial, and (3) congenital.

I. **Carcinomatous Stricture of the Intestine.**—Cancer of the intestine is second only to intussusception as a cause of intestinal obstruction. Of 669 consecutive cases of obstruction at the London Hospital in the thirteen years 1893-1905, 151 were due to cancer.

Etiology.—The primary cause of cancer is no doubt the same in the intestine as elsewhere and it is equally unknown. The disposing causes are apparently injury, chronic inflammation and irritation, stagnation of intestinal contents, and senility of the tissues. The influence of injury, inflammation, and irritation are well marked in the distribution of cancer of the intestines. In the sigmoid flexure and the rectum where the contents are solid it is much more frequent. Stagnation appears to play a part by increasing the time during which the contents can damage and irritate the intestinal wall. The contents loiter in the duodenum, caecum, sigmoid flexure, and rectum, which are all special sites for cancer. Nothnagel believes that cancer not infrequently commences in the base or scar of an ulcer, and there are at least 10 cases of this sequence in the duodenum (Letulle, Perry and Shaw, and Nattan-Larrier).

The influence of *age* is not so well marked in cancer of the intestine as in cancer elsewhere. The majority of cases occur between 40 and 65 years of age, but Lonart has collected 61 cases between 20 and 30 years, Bernoulli 37 cases under 20 years of age, and Nothnagel quotes other instances at 17, 13, 12, 11, 3½, and even 3 years of age. Maydl states that one-sixth of all cancers of the intestine occur between 30 and 40 years, and one-seventh before 30

years. Cancer must not be excluded therefore when the diagnosis of an intestinal stenosis is being considered in the case of a young adult. A specimen of colloid carcinoma of the sigmoid flexure in a boy aged 10 years was recently sent to the Pathological Department of the London Hospital by Dr. Milne, and other cases of colloid cancer of the colon at an early age have been put on record (Kanthack and Furnivall, W. A. Garrard). *Sex* appears to have little influence on the occurrence of cancer of the bowel. Of the 151 cases which caused obstruction at the London Hospital 65 were men and 86 were women.

The Pathology of Cancer of the Intestine.—Carcinoma of the intestine is nearly always primary, secondary growths are most unusual except perhaps as regards melanotic tumours (Thomson). Cancer of the intestine by extension from the stomach, gall-bladder, pancreas, and female genital organs is not so rare and is considered under Compression of the Intestines (p. 767).

Primary carcinoma of the intestine always originates in Lieberkühn's crypts and is called cylindrical- or columnar-celled carcinoma, malignant adenoma. When the epithelial elements grow rapidly and preponderate, the tumour is large, soft, brain-like in consistency, and is known as medullary or encephaloid carcinoma. In other cases the tumour undergoes colloid degeneration. Very rarely there is a well-marked cicatricial reaction of the connective-tissue matrix in a slowly growing tumour, and a hard or scirrhous carcinoma is then produced.

Situation.—Primary carcinoma is exceedingly rare in the small intestine, the great majority of the cases being in the sigmoid flexure and the rectum. The caecum and ileo-caecal valve provide a small but well-defined group. The duodenum in spite of its small extent is responsible for more than half the cancers of the small intestine (*vide* p. 577). To illustrate these points I have analysed the distribution of the 481 cases of cancer of the intestine contained in the Medical and Surgical Records of the London Hospital from 1900-1905. Nothnagel's figures are appended for comparison.

Source.	Duodenum.	Jejunum.	Ileum.	Small Intestine.	Appendix.	Cæcal and Ileo-Cæcal.	Ascending Colon.	Hepatic Flexure.	Transverse Colon.	Splenic Flexure.	Descending Colon.	Sigmoid Flexure.	Rectum.
London Hospital Med. and Surg. Registers	5	3	2	5	...	11	6	3	17	12	6	103	278
Nothnagel. Path. Inst. Wien. 1870-1893	7	...	10	...	2	23	6		80			53	162

Secondary growths in carcinoma of the intestine occur later and less

frequently than in cancer elsewhere, the prognosis after early diagnosis and excision is therefore relatively good. It is said that this statement does not apply to the rare cases of cancer of the small intestine. Metastasis is most common in the lymphatic glands, but it is nearly as frequent in the liver. After these the peritoneum, omentum, and mesentery are most often invaded, and then the lungs and kidneys. Hauser states that the ordinary and hard form of cancer of the intestine chiefly produces metastases in the liver. The colloid form on the other hand is specially apt to invade the peritoneum, while in soft cancer the lymphatic glands are the main sites of infiltration.

The Anatomical Forms of Cancer of the Intestine.—The form in which a cancer is found at an operation or an autopsy depends upon the time that it has existed and the rate at which it has grown. Those forms which produce obstruction, as a rule, come under observation early, whilst those that do not interfere with the lumen of the bowel are only seen at an advanced stage. It is of practical importance to recognise several different varieties, because the effects and symptoms produced by them are very different, and the physician must be prepared to recognise cancer of the intestine in all its protean forms. (1) The nodular variety. It is probable that all cancers commence as a small nodule in the deeper part of the intestinal mucous membrane, but at this stage they can only be discovered by accident. (2) The polypoid variety is a further stage of the last. The nodule grows towards the lumen of the bowel, and very exceptionally may become pedunculated. The mucous membrane for a time is intact over the growth, but later it is ulcerated, and the tumour fungates. Such a malignant polypus may produce an intussusception, but this is rare as compared with innocent polypi. (3) The annular or ring carcinoma is one of the commonest forms in which cancer of the intestine is found. The bowel is often puckered in as though a string had been tightly tied around it, and the actual mass of the cancer is very small. As a rule only one stricture exists, but several secondary ones may be present. The annular arrangement, which is well marked in all cancers of the bowel, is due to the cancerous infection following the lymphatics which run round the intestine to the mesentery. Some carcinomas, however, appear to have started in the annular scar of a stercoral ulcer, especially in the sigmoid flexure. These scars often surround the intestine, and at first the cancerous process is limited to the scar. The diagnosis is confused in such a case by the long history of the chronic constipation, followed by simple ulceration and stricture which preceded and produced the cancer. Even at the operation or autopsy it is often only possible to determine whether the stricture is malignant or not by the microscope. The lumen of a malignant stricture is, however, usually ulcerated. The symptoms are those of progressive stenosis of the bowel unless a foreign body suddenly become impacted, when the onset of symptoms may be very acute and sudden. (4) The cancerous process has in some cases implicated some two or three inches of the intestine and converted it into a rigid tube. The lumen is, as a rule,

ulcerated and patent. The signs of intestinal obstruction are not well marked in such cases, and the symptoms are obscure. Under Intussusception (*vide* p. 796) it is pointed out that a cancer which has produced invagination often spreads in the middle layer, and converts it into an inverted cancerous tube. (5) The papillomatous or cauliflower variety is also common. When the growth of a cancer is rapid it passes quickly through the nodular and, perhaps, the polypoid forms, and projects into the bowel as an exuberant cauliflower-like mass of friable growth. At first it is attached to a part only of the circumference of the gut, and this stage is a very favourable one for removal. The sigmoidoscope enables such tumours to be clearly seen, and their mobility ascertained as high up as the middle of the sigmoid flexure (Mummery). The actual obstruction caused by such growths is slight, and is limited to some abdominal unrest and flatulence, the wind passing freely and without much delay. Tenesmus is often present, and as the growth is nearly always ulcerated some blood and mucus are frequently passed. When the growth is situated in the lower part of the colon the patient frequently has the desire to void the discharge which has accumulated during the night directly he rises in the morning, a symptom known as "morning diarrhoea," which is very characteristic. (6) A large tumour. When a case of cancer of the intestine does not terminate early as a result of intestinal obstruction, acute peritonitis, or extensive metastases, the primary tumour becomes a large mass of new-growth, which surrounds and infiltrates the wall of the intestine, and invades the adjacent cellular tissue and neighbouring organs. It fills the lumen of the gut with a fungating, exuberant, and ulcerating mass, from which a profuse discharge is poured out. In rare cases the primary growth is of moderate size, but the major part of the tumour is formed by a large mass of infiltrated lymph-glands in the mesocolon. Such large masses of new-growth are closely simulated by accumulations of solid faeces above the stricture, or by coils of gut, matted around the growth by local peritonitis in which localised abscesses may occur. Enemas and laxatives should be employed to remove such faecal accumulations, but this is no easy matter. (7) The ulcerating and gangrenous form. All forms of cancer of the intestine ulcerate, but in some the process is so active as to merit the above description. The low vitality of the tissue which is constantly bathed in corrosive fluids swarming with micro-organisms accounts for the ulceration. This condition is indicated by a profuse and frequent diarrhoea and tenesmus. The stools contain blood and pus in large quantities, and when the process becomes gangrenous they are inexpressibly foul, and sometimes contain masses of necrotic new-growth. Cancerous cachexia is usually well marked in such cases, and is due to the loss of fluids from the raw surfaces and the absorption of the products of decomposition. It is, however, remarkable how little cachexia may be present in some very extensive cases of ulceration, and this may be attributed to the absence of lymphatics in new growths. Ulceration in an annular growth may open the passage so that symptoms of chronic stenosis are suddenly

relieved by a profuse diarrhoea, and the condition of the patient may in many ways improve.

The secondary changes and results produced by carcinoma of the intestine are as follows:—(1) Chronic stenosis of the intestine. (2) Acute dilatation of the colon above the stricture. (3) Acute dilatation of the caecum. (4) Intussusception. (5) Volvulus, bending, kinking. (6) Peritonitis, Localised, (*a*) adhesive, (*b*) suppurative. General, (*a*) perforative, (*b*) due to transudation. (7) Fistulas. (8) Ballooning of the rectum.

(1) Chronic Stenosis of the Intestine.—Cancer of the intestine is the most frequent and typical cause of chronic stenosis, the pathological changes and symptoms of which have already been fully described (pp. 705, 720). The pathological changes, which are chiefly in the intestine above the growth, are, briefly, dilatation, hypertrophy, lengthening of the intestine, inflammation, ulceration, and tension-striae in the mesentery.

(2) Acute dilatation of the colon is a very common termination to a case of cancerous stricture. Within a few hours a case which was progressing apparently favourably may become hugely distended, profuse vomiting may appear with absolute constipation, and death may follow in a day or two. At an operation or autopsy the colon is found to be so enormously distended that the longitudinal striae have disappeared, and this distension may extend to the ileum and even to the jejunum, duodenum, and stomach. The tension of the intestinal walls is so great that they readily rupture, especially at the points where patches of gangrene and softening exist. The colon, as it dilates between the layers of its mesocolon, shortens that structure and anchors the gut. The contents of the colon are usually fluid in the part adjacent to the stricture, although the upper parts often contain much gas. The causes of this sudden distension are various, but may be described as a failure of the compensation provided, by a progressive hypertrophy, for an increasing stenosis of the intestine. The lumen of the stricture has in some cases been found completely occluded by a foreign body, such as a fruit-stone, or, as in a case of my own, by tea-leaves, so that it offers an insuperable obstacle to the most powerful waves of peristalsis. In many cases acute dilatation has been precipitated by the unwise administration of purgatives. The colon, which was already loaded to the limit of its powers, has suddenly had to receive from above some pints of fluid faeces, and compensation has broken down altogether. In other cases the muscular wall of the intestine adjacent to the stricture has apparently become exhausted and paralysed, and has been distended by the active intestine above it. In some instances general peritonitis has been found originating in the patches of gangrene, and this no doubt assisted in producing the sudden distension.

(3) Acute Dilatation of the Caecum.—In this condition the caecum suddenly becomes enormously dilated, whilst the rest of the colon above the cancerous stricture is usually only moderately distended and is not tense. Gangrenous patches appear in the caecum within a few hours, and are usually referred to as stercoral ulcers. The general peritoneal

cavity is infected from these patches. In the worst cases the caecum bursts and collapses, whilst the peritoneum becomes distended with free gas and fluid faeces (pneumoperitoneum). More usually the caecum becomes adherent to neighbouring structures by recent lymph, which prevents actual extravasation until these adhesions are separated. The carcinoma is usually of the ring or annular form, and acute dilatation of the caecum is often the first sign of the presence of the cancer. Of my 6 cases 3 had been in perfectly good health until ten days, six days, and two days before admission to the hospital. The sites of the stricture in my 6 cases were 3 in the sigmoid, 2 in the splenic flexure, and 1 in the hepatic flexure. It will be seen that an undue proportion of cases is in the upper colon. The mechanism of the acute dilatation of the caecum is obscure; it cannot be due to paralysis as is usually stated, for this will not account for the tension of the walls, the pressure-gangrene, or even for the rupture of the organ. It is clear that the caecum is dilated or distended by the colon below it, which suddenly regurgitates its accumulated contents, and blows up the caecum like a child's balloon. This reflux may occur in three different ways. As the contents gradually accumulate above the stricture a time comes when they are continuous as high as the caecum, and as the colon just above the stricture is greatly hypertrophied while this has not yet occurred in the caecum, spasm of the muscular colon will lead to distension of the caecum. This, however, will not account for the absolutely acute cases; in these we must either invoke antiperistalsis, which is a normal function of the upper part of the colon, or we may suppose that whilst a forward wave of peristalsis is straining to force the accumulated contents through the stricture the contraction, which closes the colon behind the wave, is burst open, and the contents are suddenly squirted back into the caecum and distend it. The mechanism of the last explanation is similar to that of a gun which blows out its breech-block instead of propelling forward its projectile, and the devastation worked behind the gun finds its equivalent in acute dilatation of the caecum.

(4) Intussusception as a result of Cancerous Stricture of the Colon.—Of 669 cases of obstruction at the London Hospital 151 were due to cancer of the intestine, and of these 5 only produced intussusception. In 2 cases the growth was inverted, and formed the middle or returning layer. The form of the cancer may be lateral and polypoid, annular, or even tubular. In some cases the invagination leads to acute intestinal obstruction, but in the majority of cases the obstruction is partial, and the symptoms subacute or even chronic. [*Vide* p. 786.]

(5) Volvulus, Bending, and Kinking as a result of Carcinoma of the Intestine.—Volvulus is a very rare sequel; no case has been recorded at the London Hospital. Bending and kinking are said to result from the distension above the stricture combined with adhesions due to peritonitis around it, but these conditions are difficult to identify. These causes may all produce an acute termination of a chronic case of cancer.

(6) Peritonitis and Suppuration in the Cellular Tissue as a result of

Cancer of the Intestine.—Infection is produced around a cancer either by a crude perforation or transudation of micro-organisms. Infection of the retroperitoneal cellular tissues is rare, but it may occur in any part of the colon. I have seen an offensive psoas abscess point in the left groin in a case in which cancer of the sigmoid flexure was adherent to the external iliac vessels, and had produced thrombosis of the iliac vein. In another case a large hard mass in the left loin was found to be a suppurating mass of cancer connected with the descending colon. Infection of the peritoneum is much more common. It may occur suddenly as when actual perforation occurs and acute general peritonitis is produced, or gradually, in which case the peritonitis is more or less localised by adhesions. These adhesions may be local or may be universal, producing general sclerosing peritonitis. Sometimes a single band, kink, or adhesion is formed which produces obstruction, and such cases may be operated on with success. Suppuration may occur among localised adhesions, and chronic peritoneal abscesses may be produced. In the region of the caecum, especially in the aged, it is difficult to distinguish such abscesses from appendicitis. In the lower sigmoid and upper rectum in women they produce a hard, indurated mass, easily mistaken for cancer of the ovaries or pyosalpinx unless the growth can be reached from the anus. Acute general peritonitis is a common termination of cancer of the colon. As a rule a crude perforation is present. This may be in the growth, in a stercoral ulcer just above it, or in the caecum. The late Dr. Slade pointed out that a suppurating gland in the mesocolon might also produce general peritonitis. The occurrence of general peritonitis in acute dilatation of the colon and caecum has already been alluded to.

(7) Fistulas as a result of Cancer of the Intestine.—When a cancer of the intestine becomes adherent to another hollow viscus, ulceration and perforation may follow and a communication may be established. A fistula has thus been formed between the intestine and another part of the intestine, the urinary bladder, the stomach, the vagina, or in rare instances the skin. A fistula between the intestine above and below the cancer has been known to relieve the obstruction (Pollosson, Rolleston). The transverse colon may communicate with the stomach so that faecal matter is vomited and food is passed unchanged by the rectum, and in fact almost any part of the colon may communicate with any other. The bladder most commonly communicates with the sigmoid flexure, but it may also be connected to the transverse colon or the caecum. The operation of short-circuiting gives good results in the case of fistulas, which will often close after the faecal stream has been diverted.

(8) Ballooning of the rectum is very often present in cancer of the lower part of the colon. It is, however, frequently found in other conditions, such as faecal accumulation, appendicitis, pelvic tumours, peritonitis, and various neuroses. [For further remarks on this condition, *vide* p. 857.]

Symptoms.—The manifestations of cancer of the intestine are so extremely varied and diverse that their description will be based on

pathological lines, and the symptoms and signs due to the cancer itself will be mentioned first and then those due to chronic stenosis and acute obstruction will be dealt with.

Symptoms referable to the Cancer itself.—A cancer of the intestine may remain practically latent for two or three years. Thus, a medical man palpated and diagnosed a cancer of the rectum, but operative treatment was refused. Nearly three years of an active and busy life elapsed before urgent symptoms of obstruction rendered colotomy necessary, when a huge cancerous mass was found filling the pelvis. Latency is more likely when the growth is small, does not ulcerate or fungate, occupies part only of the lumen, produces few metastases, and is situated low down in the alimentary canal. Cancerous cachexia is greater the nearer the growth is to the stomach. The general symptoms of the presence of a malignant tumour are vague. They are a loss of strength, a sense of depression and abdominal discomfort, a progressive pallor and change in appearance of the face.

Pain is a prominent symptom in some cases, but it is usually due to peritonitis or obstruction. It is localised correctly when the tumour is in a fixed part of the bowel, but in the small intestine, transverse colon, or sigmoid flexure it is as a rule referred to the umbilicus. In a few cases it has been "crossed"; thus in cancer of the caecum pain has been persistently referred to the left iliac fossa. In very rare cases the pain has depended on invasion of nerve trunks such as the crural or sciatic nerves, and it is then neuralgic in character and widely diffused. In degree the pain of cancer of the intestine is not severe apart from peritonitis and obstruction. It varies from a sense of discomfort to a continuous gnawing pain or the most severe neuralgia. When the pain bears some relation to the taking of food the growth is often in the lower part of the ileum or in the caecum. When it is more severe before defecation the growth is usually in the upper part of the colon.

An abdominal *tumour* is palpable in about 40 per cent of the cases. Many parts of the bowel are inaccessible to palpation, whilst in other cases the tumour is small and covered by dilated intestine above it. When palpable the tumour may vary in size from that of a nut to a child's head, and is irregular in shape, being cylindrical, smooth, or nodular. It is solid in consistency, but as a rule resonant on percussion. It may be tender or insensitive. Tumours of the fixed portion of the intestine are as a rule immobile, but those in parts of the gut with a mesentery present the greatest mobility. These mobile tumours may, however, become fixed by adhesions, and in the most abnormal positions. Tumours of the intestine frequently disappear and reappear. They are simulated by masses of faeces, by external tumours adherent to the intestine, and by masses of intestines adherent around the growth.

When the tumour ulcerates and fungates blood, pus, mucus, and occasionally fragments of growth may be found in the dejecta. Blood is present in the motions in 20-25 per cent of cases according to R. de Bovis. Except in the rare villous form of cancer it is not large in

amount. Sometimes it appears as streaks on the faeces; in other cases it is mixed; whilst in very rare instances large haemorrhages occur. Pus is found in small quantities when the growth ulcerates, but it may, of course, be present in any other form of ulceration or abscess-formation. Mucus is common in cancer of the colon. It is described by the patient as slime, and is due to catarrh of the bowel above the growth.

In a minority of the cases the extension of the cancerous process to the peritoneum and omentum is so extensive as entirely to overshadow the primary growth. There may be ascites with nodular and irregular tumours in various parts of the abdomen. The most characteristic of these is an elongated nodular mass transversely placed in the umbilical region, and formed of the rolled-up omentum infiltrated by growth. In such cases even if signs of stenosis of the intestine exist no certain diagnosis of cancer of the intestine can be made, for the intestine may be obstructed by a secondary process of matting and infiltration. In rare cases secondary growths in the liver and lungs have been the most prominent signs of the disease, and intestinal obstruction has not appeared until near the termination of the case.

Chronic and Progressive Stenosis of the Intestine due to Cancer.—In nearly all cases of cancer of the intestine the symptoms are those of a progressive stenosis. Since the only common causes of chronic intestinal obstruction after forty years of age are faecal accumulation and cancer of the colon, the diagnosis is mainly between these two conditions. Several clinical forms of cancerous stricture of the bowel exist. In most cases the signs of stenosis appear in a previously healthy subject, and it is only as the case progresses that the cause can be ascertained. In another and smaller class of cases the symptoms due directly to the growth have been present for some months before signs of obstruction appear. Lastly, the symptoms of stenosis due to cancer may be engrafted on those due to faecal accumulation, perhaps with stercoral ulceration, which has produced the growth by chronic irritation.

Constipation is in most cases the first symptom of stenosis of the intestine. It is irregular in the early stages, but is progressive, and makes purgatives and enemas necessary. Purgatives at first give great relief, after a time they cause severe pain although they act, and eventually they precipitate acute intestinal obstruction. The onset of constipation in a previously healthy man more than forty years of age should always suggest that cancer may be present. Constipation may persist for days, weeks, or even months, and then terminate in attacks of diarrhoea either spontaneous or due to drugs, and thus the alternation of constipation and diarrhoea is established. In some cases the growth ulcerates after a time, the stenosis is relieved, and the constipation ceases, but the stools will be found to contain blood, pus, and mucus. In some cases, especially where the growth is in the lower part of the colon, persistent irritative diarrhoea and tenesmus may be present. The motions may alter in shape and become small in calibre, flattened, or like the dung of rabbits or sheep. These changes are due to spasmodic contrac-

tion of the sphincter ani excited by the irritation of the growth, which is usually low down in the colon. Presently the patient begins to suffer from attacks of *colic* accompanied by vomiting, constipation, distension of the abdomen, and borborygmi. The pain in these attacks is sometimes agonising, but as a rule it is moderate. It is sometimes correctly localised at the stenosis, but often it is diffused and radiates widely. *Vomiting* is not a prominent symptom. It occurs at irregular intervals, usually after taking solid food, and frequently is one feature only of an attack of colic. *Contracting coils of intestine* are often visible through the abdominal wall, coils of intestine rising up and hardening during the attack of colic. It is true that normal peristalsis may be observed when the abdominal wall is greatly emaciated, but the movements are slow and the coils are not distended and do not harden as they contract. In the colic of enteritis and lead poisoning the coils of intestine are not visible, and it is very uncommon in faecal accumulation. When this sign is definitely present there is little doubt that an organic obstruction is present. The position of the growth may be determined with some degree of probability by ascertaining whether the contracting coils belong to the large or to the small intestine, and in which part of the abdomen the waves terminate. The small intestine is centrally placed, assumes the well-known ladder pattern, contracts quickly and vigorously, and is as a rule less than three inches in diameter. Large intestine often bulges the flank on one or both sides, is broader, and contracts more sluggishly. It must be remembered, however, that when the intestine has become exhausted just above the cancer the waves of peristalsis may stop some distance from it.

Meteorism and Distension.—When obstruction is well established the abdomen becomes distended. As a rule the distension is greater the lower down in the canal the cancerous stricture is placed, and as cancers are usually in the sigmoid flexure or rectum distension is common in these cases. When, however, the cancer is in the duodenum or upper jejunum intestinal distension may be absent, but the stomach may be distended and extend even below the umbilicus. Local distension is sometimes seen; thus, the caecum and ascending colon may be clearly enlarged, indicating cancer at the hepatic flexure, or the right flank may be bulged out whilst the transverse colon can be clearly defined, pointing to implication of the splenic flexure. Finally, when both flanks curve outwards and the left iliac region is distended the growth is probably in the sigmoid flexure or rectum. Nothnagel has pointed out that distended colon gives a hyperresonant note in the corresponding costo-vertebral angle, and that this sign may be of use in localising the obstruction, especially if obtained on one side only. When the whole abdomen is evenly distended very little can be deduced except that the obstruction is probably in the lower colon. When the colon is enormously distended and filled with fluid faeces the condition of the abdomen may closely simulate ascites. The flanks are bulging, dull on percussion, and there is a sense of fluctuation on palpation. Succussion

can, however, nearly always be obtained when the fluid is within the intestine.

In most cases *the symptoms of acute intestinal obstruction in cancer of the intestine* supervene on those of chronic stenosis and are due to acute dilatation of the colon or caecum. It has already been pointed out, however, that compensatory hypertrophy may be so complete that practically no symptoms precede those of complete and absolute obstruction. The causes which lead to the transition from chronic stenosis to absolute obstruction have already been detailed. A foreign body may become impacted, a distended coil of gut may be twisted or kinked, or the muscular wall of the intestine may become exhausted after the administration of a violent purge. Peritonitis frequently plays an important part in the acute termination of these cases. Acute dilatation of the colon is very often, and that of the caecum nearly always, accompanied by general peritonitis due to distension ulcers. The formation of localised abscesses around a cancer may produce the final and complete obstruction, or a perforation of the colon may lead to peritonitis and so to paralysis of the intestine and absolute occlusion. In any of these circumstances the symptoms suddenly become acute ; there is continuous abdominal pain marked at intervals by severe attacks of colic. Vomiting becomes frequent and urgent and after a time feculent. The bowels are entirely closed, so that neither faeces nor flatus are passed, and turpentine enemas are returned unchanged. The abdomen rapidly becomes distended, very frequently to such a degree that separate coils cannot be distinguished. Careful examination will, however, often reveal contracting coils of gut, and this is a sure sign that a chronic stenosis has preceded the acute attack even in the absence of any history of the characteristic symptoms. When peritonitis complicates the acute attack the abdomen is tender and rigid, and there is as a rule some pyrexia. If perforation has occurred the abdomen is often evenly distended and uniformly tympanitic, and when an incision is made into it a large quantity of gas and faecal matter escape. Collapse is seldom as marked as in acute obstruction due to bands. It is late in its appearance, and is due to toxaemia resulting from absorption of the products of decomposition from the foul intestinal contents.

The Localisation of Cancer of the Intestine in the Large or Small Intestine.—The chief means of distinguishing between carcinoma in the large and small intestine will be briefly stated here. Cancer of the small intestine is latent for a longer time because the intestinal contents are fluid. When the symptoms commence the pain and vomiting are as a rule more severe and persistent, when the growth is in the small intestine, and the pain is often found to be related to the taking of food. Constipation is a much more prominent symptom of growth in the colon, while blood and mucus are seldom present in the faeces in carcinoma of the small intestine. Tenesmus, ballooning of the rectum, and faeces of small calibre as a rule point to a growth in the lower part of

the colon. In a carcinoma of the small intestine the abdominal distension is, generally speaking, centrally placed, while distension of the colon bulges one or both flanks outwards. When distended coils of intestine can be seen, and especially when they contract, it may be possible to identify them anatomically. Coils of small intestine are smaller, exceedingly tortuous or arranged in a "ladder pattern," and contract more rapidly and vigorously.

Diagnosis.—In faecal accumulation and coprostasis there is as a rule a long history of constipation, and repeated enemas bring away increasing quantities of hard, compacted scybala. Moreover, large masses of faeces may be felt in the rectum, and contracting coils of colon are seldom seen in coprostasis. When, however, chronic constipation and stercoral ulceration have preceded and produced carcinoma the diagnosis is exceedingly difficult. Hyperplastic ileo-caecal tuberculosis may closely simulate carcinoma, especially when from ulceration blood and pus appear in the motions. Tuberculosis elsewhere and the longer history of the cases may assist in a diagnosis, but as a rule diagnosis is impossible. Leube has called attention to a condition of *chronic sigmoiditis* in which the sigmoid flexure is greatly thickened and forms an elongated tumour in the left iliac region. Such cases are rare and are scarcely capable of diagnosis. Foreign bodies have also been mistaken for carcinoma of the intestine. Tumours of neighbouring organs such as the stomach, gall-bladder, uterus, and ovaries may, when they invade the bowel, be mistaken for cancer of the intestine. The history and associated symptoms of such cases will generally enable a correct diagnosis to be made.

Treatment.—When the growth is inoperable and the symptoms of obstruction are slight much may be done to alleviate the patient's condition. His diet should be such as to leave a small residue only; his attention should be directed to its complete mastication, and defective teeth should be attended to by a dentist or his food may be thoroughly minced. Intestinal decomposition and flatulence may be controlled by minute doses of calomel, β-naphthol, and salol.

When definite symptoms of obstruction are present the intestine should be opened above the carcinoma, or in certain cases it may be possible to establish an anastomosis between the intestine above and below the growth. The possibility of excision of the growth is a surgical matter, but it should always be postponed to a second operation when distension and obstruction are present.

II. **Cicatricial Strictures of the Intestine** are the result of (*a*) the contraction of scars produced by healing ulcers, or (*b*) the loss of tissue due to limited gangrene or sloughing, (*c*) in the case of syphilitic strictures and in the hyperplastic form of tuberculosis the narrowing of the lumen of the intestine is largely due to the presence of dense masses of connective tissue in the wall of the intestine. As a rule the scar is annular and surrounds the gut, and in the majority of cases bending, distortion, adhesions, or folds of mucous membrane and polypi assist in

the production of intestinal obstruction due to strictures. Cicatricial stricture of the intestine is an exceedingly rare cause of intestinal obstruction if, as is customary, strictures of the rectum are excluded. This is remarkable when the frequency and extent of many forms of ulceration are taken into account. The surgical registers of the London Hospital contain 13 examples only among 669 consecutive cases of intestinal obstruction in thirteen years.

Etiology.—Cicatricial strictures may be divided into: (*a*) Those due to some form of ulceration; (*b*) those following abdominal injury; (*c*) those which result from strangulated hernias. Those simple strictures which follow the spontaneous elimination of an intussusception are fully dealt with under that heading. The 13 cases at the London Hospital were distributed as follows:—(*a*) Ulcerative 9, that is, tuberculous 4, syphilitic 2, duodenal ulcer 1, undetermined 2; (*b*) traumatic 3; (*c*) strangulated hernia 1. Nothnagel states that for practical purposes tuberculous and stercoral ulceration are the only common causes of cicatricial stricture. A few cases are, however, due to duodenal, dysenteric, follicular, syphilitic, and typhoid ulcers.

(*a*) *Strictures due to Ulceration.*—(1) Tuberculous Strictures of the Intestine.—The tuberculous infection may attack either (1) the mucous membrane, (2) the submucous tissue, or (3) the muscular coat and subserous layer. (1) Strictures produced by tuberculous ulcers are often multiple (Handford, Voehts, Nothnagel, Rolleston, Mayo Robson); as many as seven or eight have been recorded in one case. (2) In tuberculosis of the submucous layer there is a round-celled infiltration interspersed with giant-cells in the submucous tissues. The mucous membrane itself is not ulcerated. According to Tuffier as the lesion heals and contracts it produces diaphragm-like strictures. (3) Tuberculosis of the muscular and subserous coats produces the rare chronic hyperplastic tuberculous tumours of the intestine. In these cases the muscular coat is greatly thickened owing to the deposition of dense fibrous tissue, but the chief deposit of this tissue is beneath the serous membrane, which may reach a thickness of from half to one inch. Fat- and giant-cells are found on section, and a few tubercle bacilli. The lumen of the bowel is reduced to a narrow channel which may or may not be ulcerated. The extreme hyperplasia is due either to a mixed infection or to an attenuated form of the tubercle bacillus. Conrath collected 85 cases in 1898, of which the majority were in the ileo-caecal region (tumour-like tuberculosis of the caecum), but it also occurs in the small intestine and other parts of the colon. Mr. F. S. Kidd has recently reported 3 cases in the sigmoid flexure. These strictures have frequently been mistaken for carcinoma even after excision, but it is said that the limits are more diffuse in tuberculous tumours; the infiltration disappears gradually into the neighbouring intestine and symptoms of stenosis appear later. In all forms of tuberculous stricture the neighbouring lymphatic glands are affected and the mass is adherent to the omentum or adjacent organs. The chief points in the diagnosis of a tuberculous

stricture are the presence of pulmonary tuberculosis and of tubercle bacilli in the faeces, but both are often absent, and, as Lartigau has pointed out, the infection of the intestine may be primary. Amongst Bernay's collection of 70 cases of tuberculous stricture 8 only were of the hypertrophic variety; 45 were submitted to operation and 37 recovered. In most cases the mass was resected, but in some it was excluded by a lateral anastomosis.

(2) Stercoral or decubital ulcers are due to the pressure of hard masses of faeces. They are, therefore, found most frequently in the sigmoid flexure, rectum, caecum, and the hepatic and splenic flexures of the colon. Grawitz and Nothnagel believe that they are a frequent cause of simple stricture, and are often mistaken for syphilitic and carcinomatous stenoses. Nothnagel holds that cancer frequently originates in the annular scar left by a stercoral ulcer, and he has recorded such a case and quotes another by Sir F. Treves. The condition must, however, be exceedingly rare, as no case of stenosis of the intestine due to stercoral ulceration has been recorded at the London Hospital in thirteen years.

(3) Duodenal Ulcer.—Stricture following duodenal ulcer is described on pp. 559, 563. Stricture following duodenal ulcers near the duodeno-jejunal flexure, a very rare position, gives rise to large quantities of bile in the vomit. Hochhaus has reported 3 cases of duodenal stenosis due to gall-stones: 1 close to the pylorus, 1 was in the first part of the duodenum, and 1 near the jejunum. Obstruction in these cases, however, appears to have been due rather to constricting adhesions than to true stricture.

(4) Syphilitic ulceration and stricture is common in the rectum, especially in women, but the recorded cases in the small intestine and colon are often doubtful. Two such cases have been in the London Hospital; the first, a man, aged thirty-four, under the care of Mr. Mansell Moullin in 1894, had a simple stricture in the lower ileum, for which lateral anastomosis was performed, but he did not survive. The registrar reported that the specimen appeared to be syphilitic. In the second case, a man, aged thirty, under the care of Mr. James Sherren in 1903, a gummatous mass in the jejunum was resected with 10 inches of gut, and recovery followed. (For Syphilitic Ulceration, see p. 573.)

(5) Typhoid ulcer practically never leads to stenosis of the intestine. Nothnagel cites two undoubted cases only; one by Klob, and a later one by Hochenegg. The reasons for this immunity from stricture are that the ulcers are often small and run on the long axis of the intestine, and that when the ulceration is deep and extensive the patient does not survive.

(6) Dysenteric ulceration is scarcely ever a cause of stenosis of the intestine. Woodward reported that during the American War of Secession there occurred among the United States troops 287,522 cases of dysentery, and of these 28,451 were of the chronic form. 9431 died, and 3855 of these were chronic cases, yet not a single instance of stricture

of the intestine was found. Dr. Davidson (Vol. II. Part II. p. 513), however, has seen 5 or 6 cases.

(*b*) *Stricture after Injury.*—Resection of the intestine followed by anastomosis with Murphy's button is responsible for many of these. Mr. T. H. Openshaw resected a carcinoma of the sigmoid flexure in 1902 in the London Hospital, and united the ends with a Murphy's button. In 1906 the woman, who was aged sixty-one, returned with a fibrous stricture over which a fistula had formed. Stricture may, however, follow the method of suture, and even that of lateral anastomosis. An abdominal injury, such as a kick, a blow, or the passage of a cart-wheel, may partially rupture the bowel and yet the patient may recover. Stricture is then liable to follow in from one to four months. Sir F. Treves has collected 6 cases, and I have had two under my care—one, a little girl, aged three, was run over in August 1905. The mark of the wheel crossed her abdomen and she was profoundly collapsed, but she recovered. In December 1905 she was readmitted to the London Hospital with complete intestinal obstruction of eight days' duration. The ileum was found strictured and adherent to the abdominal wall at two points about 2 inches apart, and a large aperture was present in the mesentery. The intestine ruptured whilst it was being separated, and a Paul's tube was inserted. The patient did not survive. The second case was that of an ostler, aged forty, who, after being kicked in the pit of the stomach by a horse, vomited and was laid up for several days, and had suffered from abdominal pain ever since. Five months later he was admitted to the London Hospital suffering from colic and contracting coils of small intestine. A coil of jejunum was found to be kinked, with a scar crossing it obliquely and covered by adherent omentum. Lateral anastomosis was performed, and he made a good recovery.

(*c*) *Stricture after Strangulated Hernia.*—One case only has occurred in the London Hospital in thirteen years. Sir Frederick Treves has collected 11 such cases: 9 were in the ileum and 2 in the jejunum. One case was double; the rest were, for the most part, narrow, annular strictures, but in one the bowel was contracted for an inch and a half. The symptoms of stenosis appear in from a few days to some years after an inguinal or femoral hernia has been reduced by taxis or operation. It is probable that the piece of intestine which has been injured by the neck of the sac undergoes a local annular gangrene of its mucous and submucous layers, and that the repair of this loss of tissue produces the stricture.

The Anatomy of Cicatricial Strictures.—According to Nothnagel, cicatricial stricture is much more common in the colon than in the small intestine, and Sir F. Treves gives the proportion as 6 to 1. These authorities, however, include strictures of the rectum where syphilitic, stercoral, dysenteric, and traumatic ulcers are common, and, further, apparently regard as stricture cases in which there is contraction of the lumen without symptoms. Of the 13 cases at the London Hospital, 4 were in the sigmoid flexure and 9 in the small intestine.

The length of the stricture varies within wide limits. As a rule it is short, and often appears as though a string had been tied round the intestine. Syphilitic and tuberculous strictures may form tumours some 2 or 3 inches long. Where extensive ulceration of the colon has produced stenosis the intestine may be reduced to a rigid, narrow tube for a considerable distance. The contraction of the intestinal lumen necessary to produce symptoms depends largely upon the site of the stricture. In the small intestine, where the contents are fluid, the lumen is nearly obliterated before symptoms arise, unless a foreign body becomes impacted. In the rectum and sigmoid flexure, however, where the intestinal contents are normally solid, a moderate degree of narrowing will produce obstruction.

A cicatricial stricture is, as a rule, adherent to neighbouring structures, and surrounded by peritoneal exudate and adhesions. The stricture itself is formed of connective tissue, which is usually pigmented, and a round-celled infiltration is often present. In tuberculous strictures giant-cells and tubercle bacilli are usually found, and in that of syphilitic strictures peri- and endarteritis. The changes above the stricture have been already dealt with in the General Pathology of Intestinal Obstruction (p. 705).

The *symptoms* of a cicatricial stricture are those of stenosis, and need not again be given. In the *diagnosis* of an innocent stricture of the intestine from one due to cancer, the most important points are the long duration of the case without the production of cachexia. A history of previous enteritis and ulceration, injury, or of a strangulated hernia may often be obtained. Faecal accumulation seldom gives rise to contracting coils of intestine; this is the chief point in its diagnosis from cicatricial stricture. It must, however, be remembered that faecal accumulation produces stercoral ulcers, and these, when they heal, produce annular strictures, which, according to Nothnagel, frequently become malignant, so that the symptoms of these conditions pass imperceptibly into one another, and it is often impossible to make a definite diagnosis. The *treatment* is surgical and on similar lines to that of a cancerous stricture, but for obvious reasons lateral anastomosis is more often indicated.

III. **Congenital Occlusion and Stenosis of the Intestine.**—Congenital ano-rectal stenosis and occlusions are not included in this description.

Incidence.—There were 7 cases of congenital occlusion or stenosis of the intestine in 669 consecutive cases of all forms of obstruction at the London Hospital, and 6 further specimens have been brought together in the Museum by Dr. Arthur Keith. Leichtenstern states the distribution and proportion are as follows: 75 small intestine, 10 colon, 375 at the ano-rectal junction. The 13 cases at the London Hospital were distributed as follows: jejunum 2, connected with Meckel's diverticulum 6, ileo-caecal 2—and in one of these in addition the colon terminated blindly at the hepatic flexure—upper sigmoid 1, lower sigmoid 2. The walls of this narrowed portion may be normal in structure. Such

a case at the recto-sigmoid junction, 1½ inch long, was under the care of my colleague, Mr. Hugh Rigby (see also Congenital Dilatation of the Colon, p. 840). Congenital occlusion and stenosis of the duodenum and small intestine have been described on p. 564. The strictures in connexion with Meckel's diverticulum are more often above than below that structure (*vide* p. 774).

C. Causes outside the Intestine.— **I. Anomalous Forms of Obstruction due to Peritoneal Adhesions.**—This is a group of very heterogeneous cases which, however, have this in common, that the lumen of the intestine is obstructed by some arrangement of peritoneal adhesions which does not interfere with the vessels or produce strangulation.

Etiology.—The adhesions result from adhesive peritonitis usually due to appendicitis, diseases of the pelvic organs, tuberculous peritonitis, or cancer of the intestine. Operations on these conditions, especially when much gauze has been left in or when the intestine has been extensively handled, are occasionally followed by intestinal obstruction.

Incidence.—38 cases of intestinal obstruction due to adhesions occurred at the London Hospital in thirteen years among 669 cases of all varieties. Of these twenty-four were men and fourteen women; twenty-six died—a mortality of 68·4 per cent; there were also 8 cases of kinking, five men and three women; six died—a mortality of 75 per cent.

Morbid Anatomy.—The results of adhesions may be divided into (*a*) kinking, (*b*) bending, (*c*) looping, (*d*) constriction, (*e*) matting, and (*f*) shrinking of the mesentery.

(*a*) Kinking of the Intestine.—When traction is exerted on an adhesion, band, or diverticulum which is adherent to the margin of the intestine, the bowel is kinked and its lumen is closed. The sudden traction may be due to distension of the coil of intestine or to some change in its position, or the other end of the adhesion may be attached to some cavity which has been emptied, such as the uterus, an ovarian cyst, or an abscess. The symptoms of kinking are, as a rule, those of acute intestinal obstruction, but they are not so severe and sudden in their onset as in strangulation by bands.

(*b*) Bending.—In this variety of obstruction due to adhesions the bowel is bent once or several times, and is fixed in that position. The small or large intestine may be the site of bending, and the condition may follow any of the causes of peritonitis mentioned above, such as hernia, appendicitis, salpingitis, or operations on these. The mechanical production of obstruction in bending is complex; the bowel is frequently kinked or constricted at one or more of the bends, and in addition more or less of the intestinal wall is rendered immobile by the adhesions. A case of acute intestinal obstruction, which followed ten days after an ectopic gestation had been enucleated from the broad ligament, illustrates a single bend. I found a coil of ileum adherent and acutely bent at the bottom of the pelvis; on freeing it the contents gurgled past into the

contracted gut below. The following is a good example of multiple bends. A woman reduced an umbilical hernia and was seized with symptoms of acute intestinal obstruction. I found the middle of the transverse colon bent no less than six times and fixed by matted omentum. I separated the adhesions and removed the omentum. Her bowels were freely opened next day, but she did not recover.

(*c*) Looping.—One part of the intestine may be adherent to an adjacent part, so that the bowel between them is converted into a fixed loop. The causes of this condition are glandular disease in the mesentery, the local peritonitis over an intestinal ulcer, or constriction and inflammation in a hernial sac. The loop may be open, in which case another coil of gut may pass through it. On the other hand, the aperture formed by the loop may be closed by adhesions. In this case obstruction is caused either by kinking or bending at the apex of the loop, or by the adherent walls between the coils acting as a valvular septum. Loops are usually dependent in the abdomen, but in two cases I have seen them hanging from adhesions between their extremity and the upper part of the abdominal cavity. In both cases the ascending limb was enormously dilated and hypertrophied, and the descending limb narrow and contracted. In both I freed the adhesions, and in one lateral anastomosis was performed. Both cases recovered.

(*d*) Constriction or external stricture is a condition in which the adhesions around a coil of intestine narrow its lumen. It has already been pointed out that in nearly all cases of internal stricture adhesions are present around the intestine and contribute to the stenosis. As a rule, a single constricting adhesion binds the coil of intestine down to the abdominal wall, but in one curious case a coil of ileum lying free in the peritoneal cavity was almost severed by a constricting band crossing it from the mesentery. I performed lateral anastomosis, and the woman recovered perfectly. In many cases the adhesions are multiple and irregular in their shape and direction, and produce obstruction by their cumulative effect. Leichtenstern describes a circumscribed chronic constricting peritonitis at the hepatic, splenic, and sigmoid flexures of the colon, which he regards as a secondary result of coprostasis. Nothnagel agrees with this view, but states that stercoral ulcers are usually present. The hepatic flexure is frequently constricted by adhesions due to gall-stones, cirrhosis of the liver, and corset liver; the splenic flexure by adhesions from perisplenitis, and the sigmoid flexure by those arising from the female genital organs.

(*e*) Matting.—In this form of adhesions a greater or smaller extent of the intestines is matted together in an inextricable mass. The causes of the peritonitis are those already narrated, but when the condition is universal it is usually due to tuberculous glands, chronic sclerosing peritonitis, or cancer of the intestine. It is remarkable how much of the intestine may be matted together and bound down without producing any symptom of obstruction. Indeed, this occurs in nearly every case of local and general peritonitis which recovers. Obstruction when it

supervenes is a composite and cumulative process, consisting of kinks, bends, loops, constrictions, and often twists. Sir F. Treves has pointed out that a portion of the small intestine may be matted into a definite rounded tumour, which is, however, resonant on percussion. Mr. Malcolm has recorded such a case, which resembled a broad ligament cyst, and he refers to others in which the resemblance was so close that the mass was incised or aspirated. Mr. F. S. Eve had such a case under his care in 1907. Symptoms of chronic obstruction culminated in an acute attack. A definite rounded mass of matted small intestine was found to have undergone rotation, so that the entering and leaving coils were twisted; the patient recovered after lateral anastomosis.

(*f*) Shrinking of the Mesentery. Chronic Mesenteric Peritonitis.—This is an insidious cicatricial process which may implicate the whole peritoneum, but appears to originate in the mesenteries, which shrink and shrivel until the intestine is fixed firmly to the spine. According to Virchow and Nothnagel the process originates in inflammation of the mesenteric glands, due to some intestinal irritation, and is often connected with faecal accumulation. According to Virchow it usually starts in the glands on either side of the fifth lumbar vertebra, which receive the lymph from the caecum and sigmoid flexure. The relation of meso-sigmoiditis to volvulus is referred to on p. 800. Peritonitis deformans, described by Leichtenstern and Klebs, is a similar condition, due to chronic venous engorgement in diseases of the heart, cirrhosis of the liver, and granular kidneys.

Symptoms.—The most extensive adhesions may exist without the least sign of intestinal obstruction. When the bowel is obstructed the symptoms vary within wide limits. In the majority of cases there are symptoms of chronic stenosis with visible contracting coils of intestine and partial constipation. In a number of cases these symptoms arise in attacks which are more or less spontaneously relieved, and are much influenced by diet. In other cases the symptoms come on suddenly and acutely, and rapidly progress to a fatal termination. Where the adhesions are extensive, distension is not prominent. In many cases the mass of adhesions is palpable, and forms a definite rounded mass which is resonant on percussion.

When intestinal obstruction has supervened the *treatment* is surgical. Single adhesions may be separated or divided, but the risk of rupturing the distended intestine is very great. The method of lateral anastomosis is applicable to subacute and chronic cases. Enterotomy should be reserved for very acute cases with great distension.

II. **Intestinal Obstruction due to Compression by Tumours and other Causes external to the Intestine.**—This group of cases though convenient clinically can hardly be clearly defined, but the underlying principle is that the intestine is occluded by the simple pressure of some external tumour or organ. Such cases no doubt do occur, but much more often the pressure of the tumour is complicated by adhesions and by inflammatory or malignant infiltration of the wall of the intestine.

Pathology.—The factors concerned in obstruction from compression are as follows:—The coil of intestine must, as a rule, be so placed that it may be compressed against a bony surface, such as the pelvic bones, the spine, or the ribs; the intestine is more likely to be compressed where it is fixed, either because of the absence or brevity of the mesentery, or because it is adherent so that it cannot escape. For this reason obstruction from compression occurs in the rectum, the lower part of the ileum, the duodenum, and the colon, but is rare in the transverse colon and the greater part of the small intestine.

Incidence.—There were 32 cases of compression among 669 cases of all forms of obstruction at the London Hospital; twenty-nine of these were women and only three men. Examination of the notes shews that very few of the cases were due to uncomplicated pressure, for adhesions, infiltration, or inflammation were present in most of the cases. Half the cases did not survive. Leichtenstern has collected 165 recorded cases distributed as follows: In the rectum, 60 per cent; in the lower ileum, 10 per cent; in the splenic flexure, descending colon, and sigmoid flexure together, 12 per cent; in the duodenum, 7 per cent; in the ascending colon and hepatic flexure, 6 per cent; in the middle part of the ileum, 4 per cent; and in the transverse colon, 1 per cent. Compression of the rectum in the pelvis by tumours of the female genital organs is responsible for the vast majority of the cases.

Etiology.—The rectum has been obstructed in the following ways: Cancer, fibroids, and retroversion and retroflexion of the uterus; by ovarian tumours, especially when malignant; by ectopic pregnancies, hydatids, and abscesses due to salpingitis or appendicitis when they occupy the pouch of Douglas; by vaginal pessaries; by vesical calculi and sacculi; by malignant disease of the prostate; and by tumours and abscesses arising from the pelvic bones or the sacro-iliac joint. It is stated that in faecal accumulation the obstruction is sometimes due to the descent of the loaded caecum or sigmoid flexure into the pelvis and consequent compression of the rectum. The fixed portions of the colon may be compressed by retroperitoneal sarcoma or other growths; by cystic disease, hydronephrosis, or malignant disease of the kidneys; by abscesses around the kidneys, caecum, or in the psoas sheath, or by tumours of the liver, gall-bladder, or spleen. The small intestine may be pressed upon by mesenteric cysts, tumours, glands, or hydatids, by omental cysts and tumours, by a wandering spleen or floating kidney, or, in rare instances, a coil of intestine may become caught between the liver and the ribs. The duodenum has been compressed by cysts, abscesses, and cancer of the pancreas, by glands in the small omentum, and by haemorrhage into the cellular tissue around it. The following is a good example of the latter condition: A small boy was run over across the upper abdomen. His abdomen was soft and not tender, but his stomach became greatly distended and he vomited repeatedly. I performed laparotomy and found his duodenum surrounded by a large blood clot, but the peritoneum over it was intact; the abdomen was closed, and he was

fed by the rectum for a few days until the gastric distension subsided and he was able to take food without vomiting. He made a perfect recovery.

It is stated by Patel that the intestine above a simple compression is dilated but not hypertrophied unless ulceration be present. Ulceration is, however, not infrequent in compression, especially in the rectum, and it may then produce diarrhoea. In a case under my care a large hydatid in the recto-vesical pouch pressed upon the rectum. A large faecal mass accumulated above the point of compression and produced a stercoral ulcer, some 2 or 3 inches in diameter, from which persistent diarrhoea resulted.

The symptoms present great variety. In 22 cases collected by Sir F. Treves, the symptoms were acute in 12, subacute in 2, and chronic in 8; the proportion of acute cases in this series is probably excessive. Leichtenstern states that it is far more frequent for symptoms of chronic stenosis to precede the symptoms of acute occlusion. The latter is due to sudden changes in the position of the tumour or its rapid growth; to the lodgment of a foreign body at the point of compression; to the paralysis and distension of the intestine above, or to kinks or volvulus of the dilated coils of intestine. Since strangulation does not occur the onset even of acute symptoms is, as a rule, less abrupt, and the pain and collapse are less severe than in those forms of obstruction in which the veins are compressed. The tumour responsible for the symptoms can, as a rule, be felt and its nature diagnosed.

The *treatment* is surgical. When possible the tumour should be removed or emptied, as in the case of a pregnant uterus, an abscess, or a hydatid. When this is impossible, either a fistula is established above the obstruction or an intestinal anastomosis is performed so as to short-circuit the obstruction.

Pressure on the Transverse Part of the Duodenum by the Superior Mesenteric Vessels and Nerves.—This cause of intestinal obstruction was described by Rokitansky in 1842, and subsequently by Kundrat and Snitzler. Albrecht reported 2 cases of his own and collected the published cases up to 1899. Dr. W. Ewart (1899) contributed the first paper in English, in 1900 Byron Robinson independently described the condition in America, and more recently the subject has been referred to by Kelling, Campbell Thomson, Zade, Neck, Nothnagel, Finney, and Connor, who collected 18 cases in 1906.

Morbid Anatomy.—The superior mesenteric artery arises from the aorta behind the pancreas, and issuing from the lower border of that organ crosses the duodenum transversely where it lies upon the lumbar spine. In the cadaver, if the small intestines be drawn firmly downward and backward so as to put the mesentery on the stretch, and then the gas be emptied from the stomach along the duodenum, its passage will be seen to be obstructed at this point and the duodenum will become distended. This anatomical arrangement may become of pathological importance under certain conditions. In most of the recorded cases

enteroptosis has been present, so that the greater part of the small intestines drops into the pelvis, this condition being more complete when the intestines are contracted and empty and when the abdominal wall is relaxed. According to Kundrat some peculiarity of the mesentery is necessary, and obstruction is more likely to occur when that structure is rotated, so that its sharp right margin is applied to the third part of the duodenum. A low position of the duodenum appears to expose it more to pressure by the mesentery. Several cases have been the subjects of lordosis, which rendered the spine more prominent, and the continued dorsal position appears to have contributed to the post-operative cases. In some of the cases of acute paralytic dilatation of the stomach it would appear that the small intestines have been driven downwards and backwards into the pelvis by the enlarging organ, and that the mesentery has been drawn tightly across the duodenum. In such cases, in addition to the stomach, the duodenum is distended to the point at which it is crossed by the mesentery, and a vicious circle is thus established. This cannot be accepted as the primary cause of all cases of acute dilatation of the stomach, nor does it always follow as a secondary result, for the dilatation frequently extends a variable distance into the jejunum, and profuse diarrhoea is often present in acute gastric dilatation (106). Various degrees of this condition are found. One of the pathologists at the London Hospital informs me that it is not uncommon to find dilatation and hypertrophy of the duodenum down to the point where it is crossed by the mesentery in cases in which the stomach is not much distended and no symptoms of the condition have arisen during life. Byron Robinson found this condition more or less marked in 15 to 20 of 560 autopsies, a proportion of from 3 to 4 per cent.

Symptoms.—Many of the cases have followed abdominal operation, and we may suppose that the condition is then the result of existing enteroptosis, rendered active by paresis of the stomach due to the anaesthetic, the empty condition of the small intestine, and the maintenance of the dorsal position for a long time. The onset of acute symptoms in other cases has been attributed to vomiting, straining, or even laughing. The feature of these cases is the gastric dilatation which, as a rule, produces a resonant epigastric swelling, which may be definitely identified as the stomach. The pulse is generally rapid and feeble, and the patient exceedingly weak. Snitzler recognised and cured his case.

Treatment.—The position of the patient should be so arranged as to relieve the tension on the mesentery. The genu-pectoral position has been recommended, but the patient is seldom strong enough to assume it, and should then lie on his face or right side, whilst the pelvis is elevated by a pillow and the foot of the bed is raised. The lower abdomen may be manipulated to induce the intestines to pass upwards. The stomach should be emptied by a tube and washed out, and should distension recur this may be repeated once or twice. Should these means fail, laparotomy may be performed, and various procedures, such as

manipulations, gastrotomy, and gastro-enterostomy, may be carried out; but these are desperate measures (*vide* p. 553).

III. **Internal Herniaform Incarceration of the Intestine.**—This group of cases of intestinal obstruction includes: (1) Obstruction due to bands, including Meckel's diverticulum; (2) obstruction due to apertures; (3) obstruction due to internal hernia. In all of these a similar part of the intestine is surrounded in the same way by a ring-like constriction. The symptoms are, therefore, almost identical, and the treatment is on similar lines. They are, therefore, appropriately discussed together.

(*a*) **Intestinal Obstruction due to Bands.**—*Definition.*—Bands are cord-like structures of very different origins, which may constrict a coil of intestine in several ways.

Morbid Anatomy and Pathology.—Under this heading the origin of bands will first be discussed; then the part of the intestine which is usually obstructed by them; finally, the various ways in which the obstruction is produced will be set forth. The forms of bands are as follows:—(I.) Solitary peritoneal bands; (II.) Omental bands; (III.) Meckel's diverticulum and other diverticula of the intestine; (IV.) Normal structures abnormally attached. At the London Hospital during thirteen years there were 669 cases of intestinal obstruction, among which 67 were due to bands other than Meckel's diverticulum. Of these 67, 34 were in men and 33 in women; 36 cases died—a mortality of 53·7 per cent.

(I.) *Peritoneal bands* are formed by the moulding of the plastic lymph thrown out during an attack of peritonitis. At first the intestine is paralysed, but as its motility returns the plastic exudate is drawn out and moulded into a rounded cord by the continuous peristalsis of the intestine. Only a minority of peritoneal adhesions persist, the majority are absorbed in a truly wonderful fashion, even when they have become fibrous. The persistence of inflammatory products is due to persistence of the cause. In the case of peritoneal adhesions and bands it is some such cause as a concretion in the appendix, a Fallopian tube full of pus, or a caseous tuberculous gland in the mesentery. The situation of bands is determined by the common sites of localised peritonitis. Such are the appendix region, the uterus and its appendages, the hernial rings, the lymphatic glands in the mesentery, and the site of any surgical procedure, especially if non-absorbable sutures have been buried. Ulcers of the lower ileum and the sigmoid flexure produce localised peritonitis, and malignant disease of the intestine may produce adhesions and a band. Peritoneal adhesions are not rare about the gall-bladder and stomach, but bands are seldom produced, and coils of intestine are still more rarely strangulated there. General peritonitis, whatever its origin, but especially when tuberculous, may leave behind it a solitary band among many short adhesions. The shape, size, and length of peritoneal bands vary widely. They may be a foot in length or but the fraction of an inch. Some are thick, others thin, whilst some are round and cord-like, whilst yet others are flat and ribbon-shaped. For the most part bands are attached at

both ends, but they may break at either end or near the middle. In this case they do not often snare a coil of gut unless they happen to terminate in a knob or excrescence. Although peritoneal bands are more often single, as the name "solitary" implies, they may be multiple, and it has not infrequently happened that the wrong band has been divided at an operation.

(II.) *Omental bands* are more numerous than isolated peritoneal bands, apparently because the shortest adhesion may produce an omental band, and, further, because the omentum is a normal structure and is not liable to be absorbed when the cause of the peritonitis has subsided. An omental band has one constant attachment, namely, to the transverse colon and stomach, or to the general mass of the omentum. The other end is attached to some focus of peritonitis. Omental bands are more often rounded, multiple, and arranged in a fan-shape than peritoneal bands, and they are never very short. Strangulation by them is seldom so unyielding as in the case of peritoneal bands, because the mobility of the transverse colon and stomach permits of a considerable margin of "slack." It must be emphasised that they are frequently multiple, so that when omental adhesions are divided all should be severed.

(III.) *Meckel's Diverticulum and other Diverticula of the Intestine.—Incidence.*—Meckel's diverticulum caused intestinal obstruction 21 times out of 669 cases in thirteen years at the London Hospital; 17 were men and 4 women; 16 died, giving a mortality of 76·2 per cent, that is to say, half as great again as the mortality of other forms of band (53 per cent). Meckel's diverticulum is the persistent neck of the yolk-sac which passes with the vitelline vessels from the umbilicus to the ileum at an early stage of foetal development. This structure should be obliterated in the human embryo during the sixth or seventh week of foetal life. This obliteration is, however, often arrested at various stages, and a wonderful variety of pathological curiosities is thus produced. Not all of these produce intestinal obstruction, but so many are associated with that condition that it seems advisable to describe all the varieties of vitello-intestinal vestiges. These pathological conditions may be classified as follows:—

(*a*) Diseases of the umbilicus due to the persistence of the vitello-intestinal canal. (1) An adenoma or entero-teratoma may be formed. These tumours were first described by Kolaczek in 1875, and afterwards by Kustner in 1877. They are small, pedunculated, firm and elastic, and not unlike a raspberry. They are first noticed about the third month after birth. On section they are found to be formed of Lieberkühn's crypts situated on a muscularis mucosae. The centre is composed of plain muscle-fibre, according to some arranged in longitudinal and circular layers. A dimple is often found at their apex and a small pouch of peritoneum in their base, which may contain a coil of intestine liable to be ligatured with their base. (2) Primary columnar-celled carcinoma of the umbilicus may develop even late in life (C. D. Green). (3) The vitello-intestinal canal may remain open at both ends and a congenital

faecal fistula result. In Mr. Bernard Pitts' case the fistula appeared at the third week after birth and closed spontaneously at the seventh year. Vitello-intestinal fistulas are distinguished from urachal, gastric, and biliary fistulas by the nature of the fluid which escapes. (4) Various degrees of prolapse occur when the opening of the canal at the umbilicus is large. When the canal alone prolapses there is a single orifice, but when the prolapse is more complete the upper and lower ends of the ileum appear. Faecal matter escapes from the upper orifice alone. Even further degrees of prolapse may develop, and an umbilical hernia may be associated with the condition. (5) Intussusception may occur in the ileum either above or below the canal, and project from the umbilicus. When the intussusception is above the canal, intestinal obstruction will result, but when it is below, the symptoms will be those of marasmus from a fistula of the small gut. In Mr. Golding-Bird's case, a child four weeks old, a probe could be passed all round between the intussusception and the prolapsed mucous membrane of the canal, and faecal matter escaped from the upper coil of ileum by the side of the intussusception. Reduction was impossible, and the child died in three days. (6) The canal may be closed at its deeper end, but remain open at the umbilicus and secrete succus entericus. This form of vitello-intestinal fistula may be treated by cautery or excision. (7) A cyst is formed in rare cases by the occlusion of both ends of the canal. Such cysts have been excised and their nature determined by microscopic section.

(*b*) A Meckel's diverticulum is present and is attached to the umbilicus. This is an uncommon attachment for a diverticulum. Cazin found Meckel's diverticulum attached to the umbilicus in but 3 of 23 attached cases. A. E. Halstead found the diverticulum attached to the umbilicus 15 times in 48 cases which produced obstruction. The diverticulum extends, as a rule, a part only of the way from the ileum to the umbilicus, and is then continued as a fibrous cord. The direction of the diverticulum is, as a rule, nearly vertical, because the last coils of the ileum are normally situated in the pelvis. As to the manner of strangulation of the intestine, this may be, under a band, over a band, by noosing or knotting, or, finally, the diverticulum in consequence of its attachment to the umbilicus may form a part of a congenital strangulated umbilical Littré's hernia.

(*c*) Meckel's diverticulum has broken free from the umbilicus and is unattached at one end. (1) The commonest form of Meckel's diverticulum is that in which it exists as a free thimble-like projection from the ileum, producing no symptoms. This condition is found in 2 per cent of all human beings. The diverticulum springs from the free border of the ileum. Its position in the adult varies from 12 to 52 inches from the ileo-caecal valve, and the average is about 20 inches. Mr. Bilton Pollard has described a diverticulum which originated 24 inches below the pylorus, and, after a course of 3 feet, terminated at the umbilicus. The structure of the diverticulum is that of the lower ileum. Solitary follicles, and even Peyer's patches, have been found in them. The opening

into the ileum is sometimes valvular, but more often it is the widest part of the diverticulum, and as wide as the ileum. The shape of a free Meckel's diverticulum is usually that of a finger, but it may be cylindrical, pear-shaped, or its termination may be enlarged by diverticula, so that it bears some resemblance to a hammer. (2) When a free Meckel's diverticulum is pear-shaped its cavity may become so distended as to interrupt the ileum at its point of attachment and produce intestinal obstruction. (3) Volvulus of a free Meckel's diverticulum has been recorded, produced by the distended viscus rotating on its axis. (4) The mesentery of a free diverticulum may twist, kink, or stricture the ileum where it passes across it to join the mesentery. Dr. Arthur Keith has dissected such a specimen, which is in the Museum of the London Hospital, and I have operated on such another contained in a congenital inguinal hernia. (5) Intussusception may result from a free diverticulum becoming inverted into the ileum, into which it projects as a polypoid mass. The coil of ileum below then grasps this mass and drags it down the gut, invaginating the attachment of the diverticulum above. These intussusceptions may reach, and even project through, the ileo-caecal valve. Clinically, they are usually chronic, but terminate acutely. It is an interesting problem whether the base or apex of the diverticulum is invaginated first; it is probable that the process more often commences at the base as a prolapse of the mucous membrane, which as it is drawn down the ileum inverts the diverticulum from base to apex. A similar process is seen in invagination of the vermiform appendix (see also Intussusception, p. 787). (6) A free Meckel's diverticulum may terminate in a rounded knob or ampulla. Should this band encircle a coil of intestine and become knotted the swollen end will prevent the knot from slipping. Specimens of this condition may be seen in the Royal College of Surgeons' Museum, No. 2695 B, and in the London Hospital Museum. Parise has fully described the varieties of complicated knots which may be so produced. (7) A free Meckel's diverticulum may be found in a hernial sac (Littré's hernia). I have encountered diverticula in inguinal and umbilical hernias. They have also been found in femoral hernias. In some instances they have been strangulated, and then form one variety of partial enterocele.

(*d*) The free end of a Meckel's diverticulum has formed a new attachment. This is the form of Meckel's diverticulum which most frequently produces internal strangulation, and the end is, as a rule, attached to the mesentery. Cazin collected 20 cases of this variety with the following attachments:—Near the inguinal ring, 1; to the small intestine, 6; to caecum, 2; to the colon, 1; to the mesentery, 10. The figures given by A. E. Halstead in 30 similar cases causing obstruction are:—To the mesentery, 23; to the mesocolon, 1; to the omentum, 1; to the small intestine, 3; to the mesorectum, 1; to the appendix region, 1. The diverticulum has also been found attached to the bladder, pelvic organs in the female, hernial rings, and to the abdominal wall away from the umbilicus. Localised peritonitis has determined the point of attach-

ment in many cases, and glandular disease of the mesentery plays as important a part as in omental and peritoneal bands. The fresh attachment of the diverticulum to the mesentery may be either above or below its origin from the ileum.

The length of such a diverticulum varies greatly. There are three forms: usually the lumen extends into a part only of the band, in rare cases it reaches to the end, and in still rarer instances the diverticulum is represented by a fibrous cord throughout.

Methods of Strangulation.—(*a*) As a rule, the coil of gut passes beneath the arcade formed by the adherent diverticulum. (*b*) Strangulation by noosing and snaring is liable to occur when the band is long. Most fantastic knots have been described when coils of gut passed through several apertures. (*c*) In nearly all cases the diverticulum strangles itself and becomes distended and gangrenous, so that peritonitis is an early symptom and complicates the diagnosis. I have seen a diverticulum rolled upon itself three times by the entering coil of gut, so that volvulus of both ends was produced in opposite directions. (*d*) Volvulus of the incarcerated coil of gut is not uncommon. (*e*) The ileum may be kinked or twisted at the point of origin of the diverticulum. Strangulation of Meckel's diverticulum chiefly occurs in young males, and is complicated early by peritonitis.

(*f*) A stricture of the ileum in connexion with a Meckel's diverticulum. These strictures are, as a rule, just above the origin of the diverticulum from the ileum, but they also occur below it. Their mode of origin has been much disputed, but there appear to be at least four varieties. (1) Those of congenital origin due to some irregularity of the obliterative process which normally shuts off the diverticulum from the ileum. The same obliterative process is seen in a more advanced stage at the same spot in cases of closure of the ileum by a septum of mucous membrane, or even of complete obliteration for a considerable distance (L. Hudson). (2) Traction strictures. Some have regarded these strictures as acquired and due to traction on adherent diverticula. Ulceration and cicatricial contraction are supposed to be produced thereby. (3) The mesentery of a Meckel's diverticulum is often well developed, and may then kink or constrict the ileum at the point where it crosses it. (4) The neck of a congenital umbilical hernia may narrow and nearly divide the coil of ileum above a diverticulum adherent to the umbilicus. I have added such a specimen to the London Hospital Museum. These strictures, as a rule, give rise to no symptoms, but in several instances ulceration and perforation have resulted.

(*g*) The Meckel's diverticulum has entirely disappeared, but the vitello-intestinal vessels have persisted as a fibrous cord. Such a fibrous cord passes, as a rule, from the umbilicus over the ileum to become continuous with one of the main branches of the mesenteric artery, and it is not attached to the free border of the ileum as a diverticulum always is. These bands may become detached from the umbilicus, and remain free at one end, or they may form some other attachment. In a case of this

kind, which I dissected and added to the Museum of the London Hospital, the band had deeply constricted a coil of ileum, but no actual obstruction had apparently occurred. (For other diverticula, see p. 566.)

IV. *Bands due to Pedicles and Normal Structures abnormally attached.*—The pedicles of ovarian tumours or of fibromyomas of the uterus have been known to produce strangulation of the intestine. The normal organs which may become bands are the vermiform appendix, the Fallopian tubes, the pedicle of the hydatid of Morgagni, appendices epiploicae, and the mesentery. The organ may be drawn out to an extravagant length—for example, a Fallopian tube and hydatid of Morgagni have been elongated to a length of some 6 inches, as is shewn in two specimens in the London Hospital Museum. Strangulation by a free vermiform appendix has been reported, but, as a rule, the end is adherent. The apices of two appendices epiploicae have become adherent, and a coil of intestine has passed under the arcade so formed.

Strangulation by bands nearly always occurs in the lower part of the small intestine; this is due to its dependent position in the pelvis, and because this is the common site for bands. With the exception of the sigmoid flexure, the colon is too broadly attached to be easily surrounded by a band. A portion of the lumen of the intestine may be strangulated or a loop many feet in length. Coils of intestine must frequently become incarcerated by bands and escape again, but when once the band is fairly in position many circumstances will tend to render strangulation complete. The cord is naturally prone to slip up to the neck of the coil. Whatever tends to distend the coil of gut will draw more gut and mesentery into the noose and render it tighter. The veins will be pressed upon, and the intestine will become engorged with blood and its lumen distended with blood-stained serum and gas. At an early stage of incarceration the peristaltic action of the gut above will force ordinary intestinal contents into the incarcerated coil, and these contents will less easily leave it. In these various ways the contents of the ring are progressively increased until the strangulation is tight and complete.

The various methods of strangulation of intestine by bands may be enumerated as follows:—

(1) Incarceration under a Band.—In this condition a coil of gut is incarcerated between a band and as a rule the abdominal wall. When one end of the band is attached to the mesentery the band may cross the gut once only, and the vessels are not then interfered with. When a complete knuckle of gut passes beneath the band strangulation may occur. This method of strangulation by a band is as a rule subacute, and the intestine above the obstruction has time to dilate, hypertrophy, and become visible through the abdominal wall. It is about as common a form as noosing and snaring. (2) Incarceration over a Band.—In this form the coils of intestine are slung over the band as wet clothes are slung over a clothes-line. The symptoms are subacute and comparatively mild. I have operated on such a band formed by an attachment of the

omentum to an inflamed mesenteric gland. (3) The commonest form of incarceration by bands is probably that by noosing, snaring, and knotting. The band is as a rule arranged in a simple spiral, but any knot or loop which can be formed by a piece of string attached at both ends may occur. (4) The intestine may be kinked at the point of attachment of the band in addition to being incarcerated by the band in some other way. (5) Volvulus of the Intestine Incarcerated.—The obstruction of the coil of gut incarcerated is often found to be complicated by twisting on the narrow neck formed by the encircling band. I have known this volvulus to recur and produce a second attack of obstruction although the band was entirely removed at the first operation. (6) Strangulation and Volvulus of the Band itself when it is a Diverticulum.—This is an important element in strangulation by Meckel's diverticulum, for the band here is of the same structure as the intestine, and gangrene and perforation not uncommonly result when it is twisted or one part of the band crosses another part.

The symptoms of intestinal obstruction due to bands are often more subacute than is generally supposed. Strangulation by knotting and noosing is of the most acute form of intestinal obstruction, and is indeed the type of acute strangulation. When, however, the intestine is incarcerated over or under a band, or even when it is snared by a slack cord, the symptoms may be so far subacute as to lead to procrastination from day to day with drugs and enemas before surgical aid is invoked. The symptoms of acute strangulation have already been described, but we may here briefly refer to these subacute cases which form about half those due to bands. In 68 per cent there is a history of some cause of local peritonitis, such as a hernia, appendicitis, uterine disease, or tuberculous peritonitis. The patient is usually a young adult. The onset is as sudden as in the acute form, but much less severe, and may readily be controlled by small doses of opium. Collapse is slight and often absent, vomiting is irregular and chiefly occurs after attempts to take food or after the administration of purgatives, flatus is passed per anum for the first day or two, and enemas may give a poor result. The pain does not amount to agony, but attacks of colic are more marked and are accompanied by vomiting. After a day or two distended coils of intestine become visible, and may be seen to contract and be felt to harden. There is then no doubt that a partial obstruction is present, but the gut is often seriously damaged at the point of strangulation. The difficulty of early diagnosis in such cases is very great, and I have not only operated on several cases which had been under skilled medical observation for three or four days, but in two cases I have delayed operation for a similar period. The important points appear to be the absolutely sudden onset in a patient previously in perfect health, and the continuance of vomiting and colic associated with almost complete constipation and a normal temperature, symptoms which therefore cannot be attributed to gastro-enteritis. Meckel's diverticulum when it acts as a band produces as a rule the most acute symptoms of intestinal obstruction, and signs of

general peritonitis appear early, because the diverticulum very soon becomes gangrenous.

Treatment of strangulation due to bands is entirely surgical, and should be carried out as soon as the diagnosis of obstruction has been made. The band is divided between forceps, and then cut as short as possible after ligature. A Meckel's diverticulum should be resected, and it is nearly always necessary to remove the adjacent part of the ileum with it. Hence the high mortality of this form of strangulation (76 per cent at the London Hospital).

(*b*) **Strangulation through Slits, Apertures, or Holes.**—The great majority of these cases implicate the mesentery or the omentum, and are congenital or traumatic in origin. A few are due to atrophy. There were 7 cases of apertures at the London Hospital in thirteen years out of 669 cases of intestinal obstruction; 5 were men, 2 women—6 died and 1 survived. The sites of the apertures were as follows: mesentery 3, ascending mesocolon 1, omentum 1, small omentum 1, 1 stab wound of peritoneum through which intestine passed into the subperitoneal tissues. The mesentery is the most frequent site of apertures, especially in its lower part near the caecum. Sir Frederick Treves has described a spot in this situation free from fat, lymphatics, and vessels, and surrounded by the anastomotic branch between the ileo-colic and the last mesenteric vessel. This spot is always thin and may be actually perforated. Strangulation through such an aperture has been recorded. In most cases there has been some previous injury, such as a kick or the passage of a wheel over the abdomen. I have added a specimen to the London Hospital Museum in which a large aperture had been produced in the mesentery of the lower ileum, but death was due to subsequent stricture of the intestine at two adjacent spots where the gut had been nearly completely ruptured. In the case of another specimen in the Museum of the Royal Free Hospital a surgeon whilst exploring the abdomen had in all probability passed his finger through the fatty friable mesentery. Two coils of gut had become incarcerated in the aperture, one from before backwards and the other from behind forwards. Strangulation has also been recorded through slits in the mesentery of the transverse and descending colon and even of the appendix. The omentum is the second commonest site of apertures. The majority have followed injury, but some have apparently been congenital in origin. After peritonitis the omentum may be found divided into many bands with apertures between. In other cases nearly the whole of the small intestine has protruded through a large aperture in the omentum which was reduced to a cord-like sling below it. Apertures in the adherent omentum have frequently been produced by surgeons in order to reach structures behind it, especially when operating on the pelvic viscera in women. Less common sites for apertures are in broad membranous adhesions, in the suspensory ligament of the uterus, and in the base of flat appendices epiploicae. Slits have also been produced by the adhesion of one organ to another, such as intestine to intestine, or intestine to the uterus, the broad ligament, or

the abdominal wall, especially at the hernial rings. False ligaments may be stretched parallel to such structures as the broad ligament or the utero-sacral ligament, a slit intervening. Coils of gut have become incarcerated in rents in the bladder, uterus, or rectum, or even in a coil of intestine which had been transfixed. Finally, in wounds of the abdominal wall, intestine may pass through the slit in the peritoneum and become incarcerated in the subperitoneal tissues.

The symptoms are nearly always those of the most acute form of intestinal obstruction. In only one case at the London Hospital were the symptoms subacute, and contracting coils of intestine were visible whilst flatus was passed. In this case nearly the whole of the small intestine had passed through a very large aperture in the omentum. This was the only case of obstruction due to aperture which has recovered at the London Hospital in thirteen years.

The treatment is to enlarge the aperture and reduce the strangulated intestine, which must be dealt with on its merits. The aperture should then be sewn up.

(*c*) **Strangulation by Internal Hernia.**—For practical purposes it would be well to include here all those cases of hernia in which symptoms of strangulation or incarceration are present, but no tumour is visible externally. It is customary, however, to describe a number of such cases as external hernias. For the sake of completeness these will be enumerated here but not described. *The external hernias without an external tumour in which strangulation may be produced* are—(i.) Exceedingly small external hernias through the femoral or inguinal rings, the linea alba, lineae transversae or semilunares, or Gimbernat's ligament in fat people. (ii.) Interstitial hernias at the inguinal, femoral, or umbilical rings. These may be present with or without an ordinary hernia at the same orifice. (iii.) Obturator, ischiatic, perineal, pudendal, rectal, vaginal, and lumbar hernias in all of which the external tumour is easily overlooked. Obturator hernia is the only one of these that at all commonly causes trouble. Its symptoms are as follows: fulness and tenderness on deep palpation over the inner part of Scarpa's triangle. Pain, formication, and rigidity down the inner side of the thigh. It is four times as common in women as in men, and the patients are nearly all over forty years of age. One case has occurred at the London Hospital in thirteen years. It was not diagnosed. It was reduced by laparotomy. The woman died from paralysis of the gut two days later.

True Internal Hernias.—Thirteen cases of true internal hernia have been admitted to the London Hospital in thirteen years out of 669 cases of obstruction. Four were antevesical, 3 diaphragmatic, 2 duodeno-jejunal, 2 pericaecal, 1 into Douglas's pouch, the mouth of which was constricted by adhesions, 1 in the left sigmoid fossa in a pouch formed by adhesions between the lower surface of the mesosigmoid and the iliac fossa. True internal hernias are as follows:

Subperitoneal Hernias in the Situation of the Inguinal Rings.—Hernial sacs are found projecting outwards (hernia intra-iliaca),

downwards (hernia retropubica and intrapelvica), or inwards (hernia antevesicalis) from the site of the internal inguinal ring. They are situated in the subperitoneal tissues. Some are associated with ordinary inguinal sacs. Some cases give a history of reduction *en masse*. It has been inferred therefore by some authorities that all such sacs are secondary to ordinary inguinal hernias either by repeated reduction producing pouching (hernia *en bissac*) or by reduction *en masse*. In the case of antevesical hernia at least the sac may open in the middle line; the patient may never have had an inguinal hernia and neither ring may be enlarged. Four cases of antevesical hernia have occurred at the London Hospital in thirteen years. All recovered. One was a congenital sac in the middle line, 2 were examples of reduction *en masse* and 1 of hernia *en bissac*.

Hernia Interna Vaginalis Testiculi.—In this form of hernia the testicle and vaginal pouch of peritoneum have failed to traverse the inguinal canal. The pouch is subperitoneal and lies on the psoas or iliacus muscle. The testis is, of course, absent from the scrotum.

Hernia iliaco-subfascialis was first described by Biesiadecki. The orifice of the sac is in either iliac fossa, and the sac passes through the iliac fascia and lies in contact with the iliacus muscle. Such a case was operated on by Mr. Mansell Moullin in the London Hospital. The specimen is now in the museum and shews the intimate relation of the external iliac artery to the sac. The pouch is said by some to originate from the ridge formed by the insertion of the psoas minor into the iliac fascia, by others it is attributed to the tense margin of the strong lower portion of the iliac fascia.

Hernia Duodeno-jejunalis.—In the dead body if the great omentum and transverse colon be drawn upward and the small intestine held over to the right the duodeno-jejunal flexure will in the great majority of cases be seen to be surrounded on the left by a crescentic fold, the plica duodeno-jejunalis. Between the fold and the flexure the fossa duodeno-jejunalis is usually present.

Etiology.—The duodeno-jejunal fossa is probably always developmental in origin. In practically all cases which have produced a left hernial sac the neck of that sac has been found to contain, upon the left side and above, the inferior mesenteric vein passing into the splenic vein, and, to the left and below, the left colic branch of the inferior mesenteric artery. In right duodenal hernia the neck of the sac is surrounded by the superior mesenteric artery or its continuation, the ileo-colic. The origin of these hernias would appear to be due therefore to the vascular fold or mesentery thrown up by these vessels in the peritoneum of the posterior abdominal wall (Waldeyer). Other folds and pouches even to the number of nine (Moynihan) have been described by anatomists, but they seldom if ever produce hernial sacs. They have been attributed to traction during the development of the colonic loop and the duodeno-jejunal flexure (Treitz), to fusion of the left side of the duodenum and

the descending mesocolon (Moynihan), and to the persistence of part of the meso-duodenum (Treves).

As a rule the first part of the jejunum is contained in the pouch and by its continuous movements enlarges it, drawing in more and more of the intestine. The sac spreads in the retroperitoneal tissues in the direction of least resistance. Usually it occupies the left side of the abdomen, is surrounded by the transverse and descending colon, and has its orifice directed towards the right. When the pouch passes upwards it will lie in the root of the transverse mesocolon in front of the pancreas and left kidney, and covered in front by the transverse colon and the stomach. The orifice will then be directed downwards and towards the right (hernia mesocolica). When the hernia has extended into the right side of the abdomen it will pass below the arch formed by the superior mesenteric artery and be surrounded by the ascending and transverse colon, or it may pass even further to the right beyond the ascending colon, so that the latter structure lies in front of the sac as in Brösike's case. As the hernia enlarges the duodeno-jejunal flexure is as a rule drawn into the posterior wall of the sac, so that only the returning coil is seen passing out of the orifice and strangulation is avoided. In this way, as in Neubauer's original case (1776), the whole of the small intestine may eventually occupy the sac and only the terminal coil of ileum pass through the orifice to enter the caecum. In rare instances the upper part of the jejunum may be contained in the sac, but in addition a coil of the lower ileum may enter the orifice and become strangulated, as in a case under my care.

Symptoms.—Strangulation is exceptional in duodeno-jejunal hernias, occurring in 3 only out of Leichtenstern's 42 cases. There were 2 cases at the London Hospital in thirteen years, one was incarcerated, the other strangulated. The symptoms are those due to tension produced by the sac on the peritoneum, mesenteries, stomach, colon, and inferior mesenteric vein. They are colic, dyspepsia, constipation, irregularity of the bowels, abdominal unrest, depression of spirits, and piles which bleed freely. There is a circumscribed globular swelling occupying the epigastrium and left side of the abdomen and resembling a movable cyst, but the tumour is always resonant on percussion and borborygmi are heard on auscultation.

Treatment.—The abdomen should be opened and the gut reduced. The neck of the sac should then be sewn up. The sac might be drained posteriorly. It should be remembered that the neck of the sac contains as a rule the greatly dilated inferior mesenteric vein and the left colic artery, which must not be divided when incising the neck of the sac.

Internal Hernias around the Caecum and Appendix.—There were two cases of pericaecal hernia at the London Hospital among 669 cases of intestinal obstruction; both were strangulated and died.

Anatomy.—Three folds of peritoneum are described in this region and five pouches. (1) The anterior vascular or ileo-caecal fold conveys a

terminal branch of the ileo-colic artery in front of the end of the ileum to the caecum at the root of the appendix. (2) The ileo-appendicular or bloodless fold (Treves) fills in the angle between the end of the ileum and the caecum and root of the appendix. It contains muscular fibres continuous with those of the caecum and in its margin a recurrent branch of the appendicular artery to the ileum. (3) The meso-appendix passes from the mesentery behind the ileum to the proximal half of the appendix. It contains in its free border the appendicular branches of the ileo-colic vessels. Caecal Pouches.—(1) The ileo-colic fossa when present lies between folds (1) and (2). (2) The ileo-caecal fossa between folds (2) and (3). According to Mr. Moynihan four cases of hernia into this fossa have been recorded (Tuffier, Little, and Partridge). In Tuffier's case there was a hernial sac. In those of Little and of Partridge the intestine passed through a slit in connexion with the meso-appendix and no sac was present. (3) The retro-appendicular fossa lies behind the appendix and its mesentery. In one case recorded by Snow strangulation of the ileum appears to have occurred in this fossa. (4) The retrocaecal or retrocolic fossa is usually seen when the caecum is drawn up. It is formed by incomplete fusion of the mesentery of the primitive caecum and ascending colon to the right loin. Several cases of retrocolic hernia have been recorded, but much doubt has been raised as to whether they were really into this fossa. The appendix is sometimes found in this fossa, the mouth of which may be closed (Lockwood and Rolleston) (see also p. 595). (5) The infracaecal pouch lies in the right iliac fossa just below the caecum. Its mouth looks upwards. Probably it is identical with the pouch of a hernia iliaco-subfascialis described on p. 779. A careful dissection of so-called retrocaecal hernias would probably shew that many of them were in reality into this fossa.

Hernia Intersigmoidea. — The fossa sigmoidea was first mentioned by Hensing in 1742 in a Giessen dissertation. The frequency of its occurrence has been variously estimated as from 52 per cent (Gruber) to 84 per cent (Treves). It is almost constant in babies, but is obliterated as life goes on by the chronic inflammatory changes in the mesosigmoid.

The origin of this fossa has been attributed to two vascular folds of peritoneum thrown up by the sigmoid artery (Waldeyer, Treves). Mr. Moynihan regards it as due to imperfect fusion of the primitive mesentery of the descending colon with the peritoneum of the left loin. The orifice of the fossa lies on the under surface of the root of the mesosigmoid close to the inner border of the psoas magnus. The sigmoid vessels are found in the anterior margin of the orifice. Behind the posterior wall of the fossa lie the common iliac artery at or near its bifurcation and the ureter. The depth of the fossa varies from a mere dimple to three inches. Its apex may reach to the middle of the kidney or the pancreas.

Hernia into this fossa is extraordinarily rare. Leichtenstern only mentions one case (Lawrence). Mr. F. S. Eve has described an undoubted case, the rest are doubtful.

Hernia Intra-epiploica.—Coils of intestine have been found surrounded by a sac of omentum; they are probably the contents of previously reduced inguinal hernias.

Hernia Ligamenti Uteri Lati.—Small pouches are sometimes found projecting into the substance of the broad ligament of the uterus. A specimen in the Museum of the London Hospital shews a partial enterocele of the ileum into the pouch.

Hernia into Douglas's Pouch.—I have operated on a case in which the mouth of the pouch was constricted by adhesions between the uterus and rectum. The adhesions were divided, the ileum released, and the patient recovered. Another case occurred at the London Hospital in 1906, but the patient refused operation and died.

Hernia through the Foramen of Winslow into the lesser Sac of the Peritoneum.—Eight cases of this hernia have been collected by Mr. Moynihan. In six of these the small intestine was implicated and in two the colon; seven were men and one a woman. Their ages ranged from twenty-five to forty-four years. It would appear that gross congenital abnormality in the attachment of the colon is necessary for its occurrence, for otherwise the transverse colon and mesocolon effectually intervene between the small intestine and the foramen. The symptoms are those of acute strangulation with severe pain in the epigastrium and a swelling resonant on deep percussion. In most cases the gut cannot be reduced, and enterostomy would therefore appear to be the treatment. No recorded case has survived.

Diaphragmatic Hernia.—This is stated to be the most common form of internal hernia; yet it is very rare, for it has been found but three times in thirteen years at the London Hospital in 669 cases of intestinal obstruction, and in one of these the obstruction was due to another cause. Leichtenstern has compiled a most complete account of this form of hernia based on 252 cases (1877), and according to Nothnagel later authorities are Grosser, 433 cases up to 1899, and Struppler, 500 cases up to 1901.

Anatomy.—The hernia is said to be *false* when only an orifice exists in the diaphragm and the contents lie bare in the thoracic cavity, and *true* when a sac of peritoneum exists which may or may not be covered by pleura. Leichtenstern gives the proportion as 212 cases of false hernias to 28 cases of true. From the position of the liver this hernia is much less frequent on the right side; of Leichtenstern's cases 180 were on the left side and only 37 on the right. The orifice in the diaphragm is due either to a congenital deficiency or to traumatism such as may result from stabs, falls, or crushes as when the patient has been run over. When the symptoms of diaphragmatic hernia have followed severe vomiting or the violent expulsive efforts of parturition, it is probable that the orifice in the diaphragm already existed. The acquired form is much commoner in men (Leichtenstern, 128 men to 22 women) and in those who follow laborious or dangerous occupations, namely, in sailors, soldiers, carpenters, and slaters.

The aperture may be small, or one-half the diaphragm may be missing. The hole is usually in the muscular part of the diaphragm and is more often behind. It is specially prone to occur at certain spots, (1) where the oesophagus pierces the diaphragm, (2) where a gap exists between the fibres passing to the ensiform process and those to the cartilage of the seventh rib, (3) at a similar gap posteriorly, between the attachments to the lumbar spine and the twelfth rib, and (4) where the sympathetic nerve pierces the crus of the diaphragm. In no case apparently has a hernia passed through the orifices for the vena cava or aorta. In most cases the hernia enters the left pleura, but less often it is found in the anterior or posterior mediastinum or even in the right pleura. The contents are nearly always the stomach, and then in order of frequency the transverse colon, the omentum, the small intestine, the spleen, left lobe of liver, the pancreas, and left kidney. It is always the greater curvature of the stomach which enters the orifice, so that the organ is rotated on its orifices and lesser curvature.

Symptoms.—A baby the subject of diaphragmatic hernia may be born dead from its inability to breathe. On the other hand it may live for many years with more or less definite symptoms of thoracic and abdominal disease. In such a case the left pleura has been opened for a supposed empyema with the result that intestine presented in the wound. In traumatic cases the symptoms may supervene directly after the injury, but even then they may subside and become chronic, so that life is prolonged for years. On the other hand, as in a stabbing case of Mr. Hugh M. Rigby's at the London Hospital, a long interval may elapse between the injury and the strangulation of viscera in the orifice. In its chronic form diaphragmatic hernia can be diagnosed in favourable cases on careful and repeated examinations. Leichtenstern states that a correct diagnosis was made five times in 252 cases. The thoracic signs are almost identical with those of pneumothorax. In both conditions the chest is prominent and fixed, and the heart is displaced. On auscultation breath sounds are absent and vocal fremitus is not obtained in either condition. On percussion in both the note is drum-like and tympanitic, or is metallic in character when auscultatory percussion is employed. Succussion may also be obtained in both. There are, however, differences. Pneumothorax occupies the whole pleura and is as often right as left, whilst diaphragmatic hernia occupies the lower part only of the pleura and is nearly always on the left side. In pneumothorax the signs are evenly distributed over the chest wall and undergo but slow changes. In diaphragmatic hernia they vary very greatly at different points, and may change even during a single examination. Abdominal symptoms such as dysphagia, dyspepsia, vomiting, or actual obstruction are not marked in pneumothorax, whereas they are seldom absent in diaphragmatic hernia. Moreover, the thoracic signs and symptoms will be found to vary in the latter condition with changes in the abdomen such as the taking of food, flatulent distension, or straining. Indeed, when the patient strains the abdomen may become flatter and the thorax more distended. Finally

borborygmi may be heard in the pleural cavity in diaphragmatic hernia. Special means may be employed to examine suspected cases. The chest may be auscultated whilst the patient swallows. The stomach and colon may be distended with fluids or with gas, and the effect on the thoracic condition noted. Gastrodiaphany may be practised or *x*-rays employed after the stomach or colon has been filled with a bismuth emulsion. Death may result either from the strangulation of the abdominal viscera, or from interference with the circulation and respiration due to pressure by the incarcerated organs.

The treatment when any is indicated is surgical. It is often necessary to open the thorax as well as the abdomen in order to reduce the viscera safely and close the orifice.

D. Intussusception.—John Hunter defined an intussusception as "the passing of one portion of the intestine into another, and it is commonly, I believe, from the upper passing into the lower."

Nomenclature.—In its simple form an intussusception consists of three tubes, two of which are placed within the third. In vertical section it is composed of six layers, three on either side of the central canal. From within out these layers are known as (1) *the entering*, (2) *the returning*, and (3) *the ensheathing* or *receiving* layer or *the sheath*. The two inner tubes form a polypoid mass projecting into the intestine below, and are known together as the *intussusceptum*, and the outer layer is then called the *intussuscipiens*. The end of the intussusceptum is *the apex*. The upper part of the intussusception where the entering layer passes into it is called *the neck*, and the ridge which is formed at this point by the junction of the two outer layers is known as *the collar*. In a *complete intussusception* the whole circumference of the gut is invaginated. An intussusception is said to be *lateral* or *partial* when one side only of the bowel is drawn as a cone within the ensheathing layer. This condition is, as a rule, the result of traction on the pedicle of a polypus or a swollen Peyer's patch. Partial intussusception nearly always becomes complete at a later stage. An intussusception, like a hernia, may be *reducible* or *irreducible*, *strangulated* or *incarcerated*, *inflamed* or *gangrenous*. According to their position they are defined as *enteric*, *entero-colic*, or *colic*. An intussusception is usually *single*, but in the intussusceptions of the death-agony they are more often *multiple*, and may be *ascending* or *retrograde* as well as *descending*. A *simple* intussusception is one composed of three tubes or six layers. A *compound* intussusception is formed when a simple one passes as a single mass into the coil of gut below it, thus producing a *double* intussusception with five tubes or ten layers. Even *treble* and *quadruple* invaginations have been described.

Etiology.—This subject, which appears really to be very simple, has been enshrouded by profuse and mysterious explanations. It is usually stated that some irregular or disordered contractions of the intestine, perhaps connected with inco-ordinate action of the circular and longitudinal muscular layers of the bowel, are the primary cause of invagination.

They are divided into invaginatio-spasmodica and invaginatio-paralytica, in which segments of the intestine contracted or paralysed are supposed to become invaginated into healthy segments. Nothnagel tetanised coils of rabbits' intestines with electrodes, and found that the contracted segment was ensheathed by the normal intestine below; further, having crushed a coil of intestine, he applied electrodes to the normal intestine below, with the result that the crushed portion was invaginated into the stimulated intestine. These hypotheses, surmises, and experiments probably have an important bearing on the invaginations of the dying, but have nothing to do with the vast majority of the intussusceptions of the living. Even for the invaginations of the death-agony I would offer a different explanation. On opening the abdomen of a boy whose duodenum had been ruptured by the passage of a cart-wheel I found the small intestines in vigorous peristalsis; waves of contraction were rapidly sweeping both up and down his bowel, and when they met the larger engulfed the smaller, thus producing an invagination. These invaginations were chiefly ascending. Dr. Bayliss and Prof. Starling have shewn that such irregular peristalsis can only occur when Meissner's and Auerbach's plexuses are paralysed, but the muscular wall retains its vitality, and these are exactly the conditions likely to occur just before death from meningitis and when the great abdominal plexuses have been crushed, the circumstances in which such invaginations are usually found.

For the intussusceptions of the living a much simpler and more common explanation can be given. As Dr. Bayliss and Prof. Starling have shewn, a bolus of food excites a reflex from the mucous membrane with which it is in contact, the coil of gut above it closing in a strong ring-contraction, the coil around and below it relaxes and is drawn over it by the longitudinal fibres. In this way the bolus is moved forward and stimulates a lower segment of mucous membrane, and fresh circular fibres contract behind it, so that the movement is progressive. Should a coil of intestine become so displaced as to touch the mucous membrane of the coil below and produce stimulation, it will be grasped and passed down the canal by normal peristalsis. The etiology of intussusception thus reduces itself to a consideration of the conditions under which a part of the intestine may come in contact with the inner surface of that below it and excite this normal reflex. These conditions are as follows, and include nearly all cases of intussusception in the living:—(i.) The presence of a polypus, or (ii.) of carcinoma of the intestine. (iii.) The inversion of a diverticulum. (iv.) Various forms of prolapse at the ileo-colic sphincter. (v.) The presence of a swollen and hypertrophied Peyer's patch. (vi.) Paralytic and relaxed conditions of the colon which permit of accidental invagination. (vii.) In rare cases a segment of gangrenous gut which acts as a foreign body. Indeed, it may be stated that if the apex of an intussusception be carefully examined a mechanical cause for the invagination will nearly always be found there.

Intussusception due to a polypus is rare, but the mechanism by which it is produced is obvious. The size of these slow-growing tumours

(adenoma, fibroma, lipoma, and myoma) and the history, often given in these cases, of long-standing intestinal trouble and attacks of colic indicate that as long as the tumour is covered by normal mucous membrane it only acts as a feeble stimulant to the mucous membrane with which it is in contact. When, however, the mucous membrane is thin and adherent or ulcerated, or the polypus is composed of carcinoma, the interior of the intestine is powerfully stimulated, and the intestine fiercely grasps the obstinate mass and makes violent efforts to propel it along the lumen of the bowel. The invagination is at first lateral or partial, and is opposite the attachment of the pedicle. Later the invagination is grasped as well as the polypus. Finally, when the whole circumference of the intestine is drawn in, the intussusception is complete.

Carcinoma of the colon seldom leads to intussusception unless it be at the ileo-caecal orifice. Mr. A. E. Barker has recorded a case in the upper part of the rectum, and has collected 11 others. Five such cases have occurred at the London Hospital, distributed as follows: ileum 1, ileo-caecal valve 1, sigmoid flexure 2, upper rectum 1. The projection of the tumour into the bowel below is partly due to growth and partly to the distension and propulsive efforts of the bowel above, which force the stricture downward like an os uteri.

Intussusceptions owing their mechanical origin to *the inversion of diverticula into the intestinal lumen* form a definite group. The inverted diverticulum forms a polypoid mass, and may readily excite the bowel below. The diverticula which become inverted are Meckel's diverticulum, the caecum, the appendix, and the angles of the colon. The inversion of the diverticulum may begin by a dimpling at its end. This is probably a common method of origin in the caecum, the angles of the colon, and in some cases of inversion of Meckel's diverticulum. In the appendix always, and in Meckel's diverticulum usually, the invagination commences at the base by a prolapse of the mucous membrane, probably due to spasmodic contraction of the muscular coats of the diverticulum. Many diverticula which are inverted never lead to intussusception. This is probably because they are soft and their normal mucous covering is a feeble stimulant of peristalsis in the coil below.

An Inverted Meckel's Diverticulum is the Point of Origin of an Intussusception.—The chief papers on this interesting subject are by Küttner (1898), Halstead (1902), Mr. Corner (1903), and Mr. Watson Cheyne (1904), who has collected 16 cases. As a rule the inversion commences as a prolapse of the mucous membrane into the ileum. In Hohlbeck's case the entire mucous membrane was prolapsed without inverting the diverticulum. On the other hand, the inversion may commence at the extremity by dimpling, as is shewn by a specimen (A. 2718) in the Royal College of Surgeons' Museum, in which the peripheral half only of the diverticulum is inverted into the proximal half. In 2 cases the invagination of the apex of a free diverticulum was due to the presence of an accessory pancreas there (Heller, Brunner). Of 12 cases

collected by Halstead 8 had gone on to intussusception and 4 had remained as simple inversion of the diverticulum. In 2 cases a stricture was associated with the diverticulum (Ewald, Cheyne). An inverted Meckel's diverticulum at first produces a lateral or partial intussusception, and only later a complete one.

The clinical history of these cases is often prolonged, but they frequently terminate very acutely. This form of invagination is very apt to lead to double and even triple intussusceptions. In Mr. Watson Cheyne's case a triple invagination existed. The diverticulum may advance to the ileo-colic sphincter and even through it. It is then tightly strangled. When it has become fixed at the valve a secondary ileo-caecal intussusception is often superadded.

Caecal Intussusception.—The Intussusception originates in an inverted Caecum.—This variety was first described by Mr. F. S. Eve in 1897. The inversion is always complete, and the fundus of the caecum forms the apex of the intussusceptum. When such an intussusception is reduced the caecum remains inverted after reduction of the ileo-caecal valve. It has usually been assumed that the inversion commences at the apex of the caecum by some accidental dimpling in, but from dissection of several specimens I believe that inversion of the caecum is usually a secondary and unimportant result of ileo-caecal intussusception. The valve prolapses into the ascending colon, and is grasped and sucked upwards. If it hang back and be more or less firmly attached, the loose inner wall of the caecum, which is continuous with the lower lip of the valve, is drawn in first and the caecum gradually inverted from its base. The ascending colon then, having obtained a firmer grip, draws in the valve and the ileum. The proof is, that in the cases I dissected the lips of the valve were prolapsed and pouting, and it is hard to see how this condition could have been produced if the valve were passively drawn in after the caecum.

An Intussusception originates in an inverted Vermiform Appendix.—This condition is exceedingly rare. Mr. H. F. Waterhouse recorded a case in 1898, and gave notes of 5 others. Mr. E. M. Corner brought the literature up to 1903, and Mr. Pendlebury has recently recorded another. The inversion always commences at the base by prolapse of the mucous membrane, and it is seldom complete. In Dr. Rolleston's case a concretion was fixed in the prolapsed mucous membrane, and this led him to compare this condition to that of prolapse of the ureter into the bladder when a stone is impacted in the orifice. This form of intussusception is very chronic, and in several of the cases inversion of the appendix had not gone on to intussusception. Mr. Ewen Stabb has reported a case, in a boy of three years of age, of intussusception caused by a hard mass in the wall of the caecum due to an adherent appendix perforated at its base. The case recovered.

The Intussusception originates in an Inversion of the Hepatic or Splenic Angles of the Colon.—Colic intussusceptions occur at the hepatic and splenic flexures and at the junction of the sigmoid flexure with the

rectum. In a little girl upon whom I operated the origin of the intussusception was the dimpling in of the splenic angle into the descending colon. I have seen a similar condition at the hepatic angle.

Intussusception due to various Degrees of Prolapse at the Ileo-colic Sphincter.—This is by far the commonest site for intussusception, and any explanation of this condition must explain this distribution. Leichtenstern has emphasised the similarities of the ileo-colic sphincter to the anus, and Dr. Keith and Dr. Elliott have directed attention to it lately. The last few inches of the ileum form a powerful detrusor and are closed by the tonic ileo-colic sphincter. As spasm and tenesmus at the anus produce first partial and then complete prolapse, so do they at the ileo-colic sphincter. In just the same way as piles above the sphincter ani lead to prolapse, so do the cluster of swollen Peyer's patches above the ileo-colic sphincter lead to the higher degrees of prolapse at that point in babies. Since my attention was directed to this, all the specimens of ileo-caecal intussusception that I have examined have shewn more or less prolapse through the sphincter. But whereas the prolapsed tissues at the rectum project into the open air, at the ileo-colic sphincter they project into the ascending colon. Moreover, Dr. Keith has pointed out that in mice the longitudinal bands of the colon apply the ileo-caecal orifice to the ascending colon, in the later stages of the digestion. In a condition of spasm they would apply a prolapse firmly to the orifice of the ascending colon until it was grasped in a contraction. Considering how common prolapse of the anus is in children, it is really remarkable that intussusception is so comparatively rare.

Intussusception is due to a swollen and hypertrophied Peyer's Patch.—Intussusceptions frequently originate some 3 to 6 inches above the ileo-caecal valve in a lateral dimple. In three specimens that I have dissected, the dimple was due to dragging upon a swollen Peyer's patch which was found at the apex of the intussusceptum.

Paralytic and relaxed Conditions of the Colon which permit of accidental Invagination.—I have seen two intussusceptions, lasting a month and six weeks respectively, which had both reached the anus, one protruding 4 inches from it. Both were easily reduced and then the colon was seen to form huge inert voluminous folds. I believe that in these cases the relaxed condition of the bowel permitted an accidental invagination of the caecum or ileo-colic sphincter, and that the indolent contraction of the colon increased this invagination without strangling it.

Intussusception due to a Gangrenous Segment of Intestine which acted as a Foreign Body.—A woman, aged forty-nine, was admitted to the London Hospital with a strangulated umbilical hernia, which was reduced. She passed blood from her anus and became collapsed. Mr. James Sherren, therefore, opened her abdomen and found the central part of her transverse colon gangrenous; as her condition did not permit of a resection her abdomen was closed. She rallied, but the vomiting continued for seven days. Diarrhoea set in and continued for three weeks. Nine days after the operation she passed a tubular slough 18½ inches long, smooth on

its inner side and rough and greyish-green outside. Eleven days after operation she passed 3½ inches of colon with the mesentery attached and the longitudinal bands visible. She then recovered, but a stenosis subsequently formed, for which ileo-colostomy was successfully performed. In this case a gangrenous piece of intestine acted as a foreign body and was invaginated. The returning layer underwent necrosis first, so that the slough was inverted. Finally stenosis followed, as is so often the case after spontaneous elimination of the intussusceptum.

The Method of Growth in an Intussusception.—In simple ileo-colic intussusception the ileo-caecal valve remains fixed, whilst more and more ileum is prolapsed through its orifice into the caecum. The process is limited by the strain on the mesentery and by the orifice becoming choked by the swollen ileum and its mesentery. This form of intussusception would be more correctly described as an extensive and complete prolapse of the ileum into the colon. Growth takes place entirely at the expense of the entering layer. Very frequently a secondary ileo-caecal invagination is superadded. In all other forms of intussusception growth occurs entirely by additions from the sheath to the middle layer. The apex remains a fixed point held up by the entering layer and its mesentery. The sheath, which is the only active layer, grasps the intussusceptum firmly at the neck and then endeavours to force it down the intestine by drawing the sheath up over it, and by downward waves of peristalsis which slide the returning layer down upon the fixed axis of the mesentery and throw it into numerous folds. As the returning layer is driven down it draws more and more of the sheath over the collar into the returning layer, and this fresh addition to the intussusceptum is grasped and driven down and compressed into folds at the next paroxysm of peristalsis. Thus it comes about that in most cases the entering layer and sheath are straight, but that the returning layer is folded and creased. As John Hunter long ago pointed out, 12 inches of intestine will form an intussusception 3 inches long; 3 inches will form the entering layer, 3 the ensheathing layer, and 6 inches will be folded up in the returning layer. Thus, in the ordinary ileo-caecal form of intussusception the ileo-caecal valve remains at the apex of the intussusceptum, and the colon is progressively folded up on the termination of the ileum as an axis, so that by the time the descending colon forms the sheath, the caecum, ascending and transverse colon are packed away in the returning layer. The great mobility of the colon in infants will even permit the ileo-caecal valve to hang from the anus, where it may be recognised by the orifice of the appendix.

Forms of Intussusception.—Intussusceptions are divided into enteric, entero-colic, and colic, according to their position. The enteric may be further subdivided into duodenal, jejunal, and ilean. The colic invaginations are the result of the passage of the ascending into the transverse colon, of the transverse into the descending, of the descending into the sigmoid flexure, or of the latter into the rectum. The entero-colic group requires more detailed subdivision, into (1) ileo-caecal, (2) ileo-colic,

(3) caecal, (4) ilean ileo-colic, and (5) the very rare appendicular. In ileo-caecal intussusception the ileo-caecal valve forms the apex of the intussusceptum: in the ileo-colic form a longer or shorter piece of the ileum is prolapsed through the valve; in caecal intussusception the inverted caecum forms the apex of the intussusceptum, and the ileo-caecal valve is laterally placed on the intussusceptum some distance from the apex. In the form known as ilean ileo-colic the invagination commences an inch or two above the ileo-caecal valve, usually, I believe, in a swollen Peyer's patch. When the intussusceptum reaches the valve it may either thrust it into the colon in front of it, producing a secondary ileo-caecal intussusception, or it may entirely or partially prolapse through the valve and then produce a like result. The relative frequency and importance of these subvarieties of entero-colic intussusceptions have been much debated lately. Sir F. Treves, Mr. Sargent, and Mr. Eccles regarded the ileo-caecal as much more common, but more recent writers (Corner, Wallace, Fagge) state that true ileo-caecal intussusception is less common than was formerly supposed. This is largely a matter of words and degree. The term "ileo-caecal" has been used in two senses to denote (1) any (entero-colic) intussusception in the ileo-caecal region, and (2) a special form in which the ileo-caecal valve heads the intussusceptum. On the other hand, true ileo-caecal intussusceptions can scarcely occur at all unless there is some degree of prolapse through the valve which can be grasped by the ascending colon. It is then a question of degree when this prolapse deserves the name of ileo-colic. The older surgeons did not distinguish subvarieties, and their "ileo-caecal" cases vitiate all the older statistics, whilst some of the younger surgeons are inclined to regard even a slight prolapse as indicating an "ileo-colic" intussusception. Mr. Wallace's small though reliable series of 19 entero-colic intussusceptions included 10 single, of which 8 were ileo-caecal and 2 were caecal, and 9 double, of which 5 were ileo-colic and ileo-caecal and 4 were enteric and ileo-caecal. Since 1900 the Registrars of the London Hospital have distinguished the subvarieties of entero-colic intussusception in 92 cases. These were distributed as follows:—Ileo-caecal 51, ileo-colic 20, caecal 21.

Multiple Intussusceptions in the Living.—More than one inflamed adherent and obstructive intussusception may be found in the same case, but this is very rare; Mr. D'Arcy Power's case in a male baby five months old shewed, in addition to an ileo-caecal invagination 2 inches long, a retrograde intussusception of the transverse into the ascending colon; both were adherent and covered by lymph. Mr. Power referred to two other recorded cases (Peregrine, Handfield Jones), and a further case has occurred at the London Hospital.

Invaginations of the Death-agony.—In the abdomen of children dying of meningitis, peritonitis, or abdominal injuries several intussusceptions are frequently found in the small intestine. They are usually retrograde in character, very short, and are neither adherent nor inflamed, so that they are readily reduced. There is no evidence of obstruction above

them, and they have not produced any symptoms. Indeed, they are due to the tumultuous waves of peristalsis which may immediately precede death in such case. In a few instances they have been found in association with other causes of obstruction, such as a faecal tumour in the sigmoid flexure or old adhesions from past peritonitis (see also p. 785).

Ascending or Retrograde Intussusceptions.—Intussusceptions during life are nearly always descending. Leichtenstern collected 593 cases, and of these 8 only were ascending. He is dissatisfied even with these 8, for he states that in all peritonitis was present, and they occurred under conditions in which retroperistalsis was already established. Retrograde intussusception, however, could scarcely originate in any other way than by a reversal of the peristaltic waves, and it is interesting that so many of the authentic cases have occurred in the colon in the region of normal antiperistalsis. Thus, Besnier has reported a case of a simple invagination of the sigmoid into the descending colon, which was adherent and of nine days' duration. Mr. D'Arcy Power has recorded one of the transverse into the ascending colon, and Jones an invagination of the descending into the transverse colon which survived eight weeks. On the other hand, Hektoen had a case in which four ascending invaginations were found in the ileum, and Mr. Hugh Rigby encountered a gangrenous retrograde invagination in the ileum at the London Hospital. If descending intussusceptions be due to the swallowing of one part of the gut by that below it, as Mr. D'Arcy Power has said, then the ascending form may be described as due to the vomiting of part of the bowel through that above it (see John Hunter on Emetics and Retroperistalsis).

Double, Triple, Quadruple, and Compound Intussusceptions.—The layers of a simple intussusception may be so firmly fixed together that the entire mass acts as one foreign body, and is ensheathed by the gut below. In this case a double intussusception is produced, composed of five tubes or ten layers. The same process may be repeated twice or even thrice. The double and triple forms occur in the intussusceptions of the dying. It is also not uncommon when an inverted Meckel's diverticulum is the origin of the intussusception. Mr. Watson Cheyne has reported a triple intussusception due to an inverted diverticulum with a congenital stricture below it. When an enteric intussusception reaches the ileo-caecal valve it may either be arrested or pass a certain distance through it. In either event the valve is often invaginated into the colon, and a secondary ileo-caecal intussusception is superadded. In some cases of double invagination it would appear that the ensheathing coat has simply become folded or wrinkled upon the intussusceptum; the secondary intussusception is often then retrograde in direction. A good example of this condition has been figured by Mr. D'Arcy Power. Finally, it is said that the coil of intestine above may become invaginated into the entering layer, and in this way produce a double intussusception. In some cases other coils of intestine or omentum are caught between the neck and collar of an intussusception and drawn into the gap between the entering and returning layers. In a specimen shewn by Mr. D'Arcy

Power (for Mr. Lucy) three feet of the upper part of the ileum were strangled between the entering and returning layers of an ilean intussusception lower down in the intestine. In an entero-colic case of Mr. H. Rigby's the lower angle of the duodenum was caught in a like manner, but was not strangulated; Prof. Delépine reported an almost identical case.

Incidence.—Of all causes of intestinal obstruction invaginations are by far the most common, and this is especially so in children. Out of 669 cases of obstruction at the London Hospital from 1893 to 1905 no less than 189, or 28·2 per cent, were due to intussusception. According to Leichtenstern half of the 473 cases he collected were less than 10 years old, and one-fourth were less than 1 year old. Consecutive series give even a higher proportion. Thus, Mr. Eccles, from an analysis of the St. Bartholomew's Hospital Registers, found that 27 of 40 cases were under 12 months of age (68 per cent), and 37 of 40 cases under 10 years old (92 per cent).

Age.—The following is the distribution according to age of 187 consecutive cases of intussusception at the London Hospital:—

Age . . .	0-1	1-10	10-20	20-30	30-40	40-50	50-60	60-70	70-80	
No. of Cases .	135	30	3	4	4	6	2	2	1	=187

In other words, 72 per cent were less than 1 year old, and no less than 88 per cent were under 10. The residue of 12 per cent was fairly evenly distributed through the remaining years of life, during which intussusception is a rare cause of obstruction. Only 4 of the 135 babies were less than 3 months old. These figures agree very nearly with those obtained from St. Bartholomew's Hospital, and are probably much nearer the truth than series of published cases such as Leichtenstern has collected.

Sex.—Intussusception is more common in males. At the London Hospital there were 131 males to 58 females; about 70 per cent were males. Intussusception at and about the ileo-caecal valve is far more frequent in children (70 per cent, Leichtenstern), but in adults enteric invaginations are nearly as common. Leichtenstern's figures for all ages are as follows: enteric, 30 per cent; entero-colic, 52 per cent; colic, 18 per cent. Once again, Leichtenstern's deductions are vitiated by the source of his figures. The London Hospital records of 110 consecutive cases of all ages shew that enteric and colic invaginations are much rarer. These cases were distributed as follows: enteric, 10 cases, 9 per cent; entero-colic, 92 cases, 84 per cent; colic, 8 cases, 7 per cent. Of the entero-colic cases 1 was due to carcinoma of the ileo-caecal valve. Of the colic invaginations 1 arose from a myxoma and 1 from a carcinoma. Of the enteric intussusceptions 2 originated in myomatous polypi, 1 in a carcinoma, 1 in an inverted Meckel's diverticulum, and 1 was retrograde. Enteric intussusceptions occur most often in the lower ileum. Colic invaginations are commoner in the descending colon and sigmoid flexure.

Pathological Changes.—*The mesentery and mesocolon*, which are drawn in between the entering and returning layers of an intussusception, are

subjected to tension, and very frequently to torsion and strangulation. It has already been explained that the mesentery of the entering layer forms an axis upon which the returning and ensheathing layers are piled up. The tension on this axis necessarily becomes greater and greater, so that the intussusception becomes curved towards the mesentery. This curvature is greatest in the entering layer, and a deep crease is often produced upon its concave side. The drag of this taut axis upon one lip of the orifice of the intussusceptum reduces this to a vertical slit directed eccentrically against the mesenteric wall of the sheath. These tension effects are only constantly seen in entero-colic intussusceptions. They are not always found in colic and enteric intussusceptions, and rectal invaginations are stated to be always straight. In advanced entero-colic intussusceptions, in which the valve hangs from the anus, several curious points arise in connexion with the mesentery and mesocolon. In the first place, it is difficult to understand how the short piece of mesentery belonging to the last three or four inches of the ileum can permit the ileo-caecal valve to traverse the colon from end to end, and even project from the anus. It must, however, be remembered that this portion of mesentery is attached about the promontory at the centre of the abdomen. From this centre the mesentery swings round like the hand of a clock, and since the length of the entering layer is not increased, no extra length of mesentery is required to reach the intussusception as it travels along the colon around this centre. Very little attention has been paid to the changes in the mesocolon which contribute to this curious phenomenon. The mesocolon in infants is extraordinarily loose and long. This permits each part of the colon in turn to be drawn towards the promontory, around which the intussusception travels in quite a small circle until it passes vertically down the rectum and out of the anus, following a course like ? or a shepherd's hook. When the process is complete the aperture within the hook is not larger than a shilling, and leads into a large hernial sac, which contains the upper part of the small intestine. The ileum issues through the hook to enter the intussusception. The anterior boundary of this hernial sac is formed by the mesocolons of the ascending, transverse, and descending colons and the sigmoid flexure, which have been drawn inwards towards the ring around the promontory. The final arrangement is not unlike a Japanese fan, in which the handle represents the mesentery of the ileum, and the periphery is attached around the circumference of the abdomen, whilst the entrance to the hernial sac is to the right of the handle where it joins the fan. Moreover, it is obvious that as the ileo-caecal valve travels round the circumference of the circle it must produce one complete twist of the mesentery of the ileum, which is fixed at its other end to the promontory of the sacrum. Indeed, the ileo-caecal valve is sometimes twisted more than once, and it then communicates these torsions to the mesentery, mesocolon, and to the intussusceptum.

The great cause of the obstruction and strangulation of an intussusception is the active and vital contraction of the sheath, and especially at

the collar. It must never be forgotten that the sheath regards the intussusceptum as a foreign body and as a violent stimulus to its lining mucous membrane. The sheath, therefore, is always endeavouring to establish a ring-contraction above the intussusceptum and to squeeze it down the intestine. John Hunter pointed this out long ago, deducing it from the deep impression made by the appendix on the entering layer. Moreover, in that chronic form of intussusception, in which the colon is voluminous, languid, and paralytic, neither obstruction nor strangulation occurs. In such a case I have seen a child pass a motion from an ileo-caecal valve hanging from the anus, and I have afterwards reduced, without the slightest difficulty, the intussusception, which had existed six weeks. Other causes of obstruction which have been given are the curving, kinking, and torsion of the intussusceptum, and the great swelling which results from strangulation. Moreover, the orifice is slit-like and applied to the side of the sheath.

An acute intussusception rapidly tends to become irreducible. [*Vide* p. 799.] The chief cause of irreducibility is the swelling, stiffening, and thickening of the layers of the intussusceptum due to oedema and extravasation of blood, the results of strangulation. These changes are best marked at the apex of the intussusceptum, and it is therefore usually the last few inches which are irreducible. Reduction is very often impossible when an enteric or ileo-colic intussusception has passed through the ileo-caecal valve and is tightly strangled there. In subacute and chronic cases adhesions between the entering and returning layers may prevent reduction, but most chronic intussusceptions are neither strangled nor inflamed, and therefore are not adherent. Torsion of the intussusceptum is a rare cause of irreducibility.

In acute intussusception *the gut above the obstruction* is little altered, except that it is dilated and congested if the case be of more than two days' duration. In chronic cases it may undergo hypertrophy and those other changes which have been described under chronic stenosis (p. 705). Very rarely perforation has occurred above the intussusception.

The intussuscipiens seldom shews gross changes. In chronic cases it is sometimes thickened, and in acute cases which have terminated fatally it is not uncommon to find patches of ulceration and gangrene opposite the point where the apex of the intussusceptum presses against it. The intussusceptum may actually project through a rent in the sheath into the peritoneal cavity. The intussuscipiens is sometimes creased and folded, and when one of these folds is very large a double intussusception is produced. If the fold project upward beneath the upper part of the sheath, the secondary intussusception is, as in Mr. D'Arcy Power's case, retrograde. If the lower part of the sheath is folded outside the upper, then the secondary intussusception is descending.

The pathological changes in the intussusceptum are the most important in an invagination. The veins of the mesentery and mesocolon are compressed by the sheath, especially at the neck, in acute cases, and as a result the intussusceptum becomes engorged, oedematous, swollen, and

finally gangrenous. The microscopic changes are those of oedema and profuse extravasation of blood into the cellular tissue from the ruptured capillaries, as in all strangled tissues. The swelling is most marked at the apex and along the convexity of the intussusceptum, apparently because these parts are free from pressure and are most peripheral. The middle cylinder is, as a rule, more damaged than the central one, which is usually narrow and compressed. In acute cases gangrene may appear as early as the third day, and those cases which survive pass the sloughs from ten days to three weeks from the acute onset.

The Spontaneous Elimination of the Intussusceptum.—The gangrenous bowel may be passed in small fragments or in extensive and complete segments of the intestine exhibiting clearly such natural features as the mesentery, the longitudinal bands, the appendices epiploicae, the vermiform appendix, or a Meckel's diverticulum. Cruveilhier has described such a specimen 3 metres long, and Mr. J. Sherren has reported a case from the London Hospital in which two sloughs, portions of the transverse colon, were passed 18½ inches and 3½ inches long respectively; the larger was inverted (*vide* p. 788). When the inner and middle cylinders are adherent, the slough is passed with the same relation of parts as in the intussusceptum. When the middle layer separates first, the portion of gut has its peritoneum outside, as in the normal state; but when the entering layer gives way first, the bowel becomes inverted and is passed with its mucous membrane outside (Treves).

In an acutely strangulated intussusception the gangrenous process is most advanced at the neck where the collar cuts into it, and this is why large sloughs separate. In a chronic intussusception, on the other hand, the process is rather one of ulceration, and is located at the apex of the intussusceptum, from which it spreads upwards. According to Leichtenstern, 40 per cent of cases of spontaneous elimination do not recover. In some, extravasation occurs around the neck, because the adhesions are incomplete. In others the separated intussusceptum obstructs the bowel lower down, and the adhesions give way. Some die of haemorrhage during separation. In others ulceration follows and carries the patient off by diarrhoea and exhaustion, or more swiftly by perforation. Occasionally the stump of the intussusceptum has led to a fresh and fatal intussusception, or a stricture has formed at the line of separation, as occurred in Mr. Sherren's case already referred to. Leichtenstern states that spontaneous elimination occurred in 42 per cent of his series of recorded cases of intussusception. Spontaneous elimination occurred once in 68 consecutive cases at St. Bartholomew's Hospital, and once in 189 consecutive cases at the London Hospital. The true incidence is therefore less than 1 per cent. Leichtenstern states that spontaneous elimination occurs in 61 per cent of enteric invaginations, in 28 per cent of colic, and in only 20 per cent of ileo-caecal intussusceptions. It is very rare in babies and uncommon before six years of age, from which time onwards about 40 per cent of intussusceptions undergo spontaneous elimination. Most of the sloughs

are passed from seven days to four weeks after the onset. When the slough is separating, the motions become extremely foul and stained with blood. In some cases profuse diarrhoea heralds the appearance of the slough, in others constipation. The older the patient the less likely is he to recover after spontaneous elimination.

Sepsis and Infection. — Those changes which have already been described under the general heading of strangulated intestine occur in an intussusception. After about two days or less, micro-organisms pass into the tissues of the invagination and even through them to the peritoneum. Inflammation, peritonitis, sapraemia, and even septicaemia are the results, so that even when an intussusception has been successfully reduced, a number of patients cannot recover because of the advanced stage to which these infective processes have already attained.

Carcinoma of the intestine which has produced a chronic invagination tends to infiltrate the returning rather than the entering layer. In a specimen in the museum of the London Hospital the returning layer is almost entirely formed of an inverted cylinder of new growth originating in the ileo-caecal valve.

Symptoms.—Nearly three-quarters of the cases occur during the first year of life, forming a well-marked clinical group with regular and characteristic symptoms. The remaining quarter of the cases is distributed over the rest of life; they are rare, occur unexpectedly, and are most irregular in their symptoms, so that it is impossible to draw an inclusive clinical picture of them. It is therefore advisable to describe first the definite clinical picture presented by intussusception in babies, and then to describe the chronic form usually seen in older cases.

Acute Intussusception in Babies. — A baby who has previously been in good health, and is often a very fine child (Hunter), and more frequently a boy, is suddenly seized with the most violent intestinal colic. The tiny sufferer draws up its knees as the attacks come on and screams vigorously, whilst its face appears to exhibit pain and astonishment. Soon after the onset of the pain the child vomits once or twice, but the vomiting is not urgent and may soon remit. Another early symptom is tenesmus. The baby strains almost incessantly, and passes small motions of blood and slime at frequent intervals. An abdominal tumour will be found in 75 per cent of the cases if an anaesthetic be given. The tumour is most frequently found in the left loin, but may be felt at any point in the colon accessible to palpation. The tumour is sausage-shaped, and about an inch in diameter and of variable length; it hardens and becomes more definite during the spasms of pain, but may nearly disappear in the intervals. There is no abdominal distension during the first two or three days, and its appearance is of bad prognosis. The rigidity and tenderness of the abdominal wall are difficult to define. As a rule the child resists attempts to handle the tumour, and these appear to excite attacks of spasm; but as the little sufferer becomes more and more exhausted, it resists manipulation less and less. In about a quarter of the cases the apex of the intussusceptum may

be felt per rectum, and it is said to resemble the os uteri. It may even project from the anus, but this is not so common as in chronic cases. The progress is from bad to worse. The attacks of pain work up to a climax after some hours, when the pain is continuous and the attacks occur as exacerbations. The tenesmus becomes more and more frequent, vomiting is variable, soon the child becomes quiet, collapsed, and ringed around the eyes, and after two days distension begins, and the temperature frequently rises, and the rigidity and tenderness of peritonitis appear. If unrelieved, the child dies from the second to the seventh day. In Mr. M'Adam Eccles' analysis of the symptoms in 40 consecutive cases of intussusception at St. Bartholomew's Hospital, 26 shewed abdominal pain, vomiting, and the passage of blood and mucus; 34 passed blood and mucus, and 34 vomited; in 32 cases a tumour was found, and in 10 it was felt by rectum; pain was absent in 5 cases; blood and mucus were not observed in 3 cases. The above description applies to practically all cases under one year old. They are almost always acute and of the entero-colic variety.

The Differential Diagnosis between Acute Intussusception and Colitis in Babies.—The only condition which closely resembles acute intussusception in babies is acute colitis. This resemblance is not surprising, for the colon is acutely inflamed in intussusception, but in addition the intestine is completely obstructed. These two conditions are alike in that they both attack babies, and usually before they are one year old; they both are frequently preceded by slight alimentary disturbance, such as vomiting and diarrhoea; blood and mucus are passed with tenesmus in both, and the collapse is identical in both. Stress has been laid on the presence of fever in colitis by some writers, but it often occurs early in the course of intussusception. Even the presence, or absence, of a sausage-shaped tumour may be fallacious, for it is admittedly not felt in a quarter of the cases of intussusception, while the spastic sigmoid flexure is often palpable in the left iliac fossa in colitis; it is, however, no thicker than the little finger, and does not harden and soften so obviously as an intussusception. The crucial point in the diagnosis is whether complete intestinal obstruction is present or not. This is ascertained by searching for bile mixed with the blood and mucus on the napkin of the baby. If it is present after the first motion or two, the case is one of colitis, or a chronic intussusception. If it is absent, the baby is suffering from acute intussusception. The two sisters of the Children's Wards at the London Hospital have observed this sign in 48 cases. In 43 cases of intussusception there was no bile on the napkin in 36. It was present in 5 cases; of these 4 were chronic intussusceptions, and in 1 case the sign failed. In 2 cases the bowels were not open after admission to the hospital. In 5 cases of acute colitis admitted as intussusceptions, bile was present in 4, and was not observed in 1 case, but at the autopsy on this case bile was found all along the colon. The second important point in diagnosis is the character of the onset. In acute intussusception this is almost always absolutely sudden. In acute colitis the onset is

as a rule more gradual and is led up to by some vomiting and diarrhoea, but unfortunately a few cases of intussusception complicate gastro-enteritis.

In intussusception after the first year the symptoms are very variable. The cases may, however, be divided into acute or subacute, chronic, and chronic cases with an acute termination. The acute cases do not differ very greatly from the above description, with due allowances for the age, and with the reservation that in older children and adults the symptoms tend to be subacute.

The symptoms of chronic intussusception are often obscure and misleading; out of 55 cases collected by Rafinesque no less than 27 were incorrectly diagnosed. The condition has been mistaken for chronic colitis, cancer of the colon, faecal accumulation, tuberculous peritonitis, dyspepsia, gastro-enteritis, wandering spleen, polypus and prolapse of the rectum, and many other conditions. There is no symptom which may not be absent or modified. A careful review, however, of each case will, as a rule, supply data on which to found a probable diagnosis. Chronic intussusception may last a month or go on for a year or more. In about 30 per cent of the cases the onset is acute, the symptoms then being alleviated and running a chronic course; but most cases come on obscurely and in a progressive fashion, the earliest manifestations being transient attacks of colic and irregularity of the bowels. The important feature of nearly all cases is the occurrence of abdominal attacks at uncertain intervals; they are often brought on by food and aperients, and as the case progresses appear at shorter intervals and become more severe. They are of the nature of colic, and are very commonly accompanied by alterations in the character of the stools. In entero-colic or colic intussusception mucus with a little blood is passed at frequent intervals during the attacks; while in enteric invagination the bowels are more often constipated. The tumour, which is palpable in about half the cases, becomes harder, larger, and more obvious when spasm is present and moves along the colon. Thus, in a case on which I operated, due to a myxoma in the left half of the transverse colon, it was only during the attacks that the tumour could be felt in the left loin, for when the attack subsided the tumour retreated beneath the left costal margin. When the pain is severe, vomiting is frequently present, especially in enteric or enterocolic intussusception. Since chronic intussusception is a form of stenosis of the bowel, dilated and contracting coils of intestine are seen in most cases and especially during attacks. Emaciation and anaemia result because of the incessant pain, diarrhoea, and vomiting, and because food, since it often precipitates an attack, is avoided.

Cases of chronic intussusception eventually become acute, perforate, or reach the rectum and anus. When they terminate acutely the patient vomits profusely, becomes absolutely constipated, and often sinks in an hour or two. Perforation occurred 22 times in 55 cases collected by Rafinesque, 1 was some distance above the intussusception, 3 were just above it; in 15 cases the sheath was perforated with hernia of the

intussusception, in 3 cases without. In one case the sheath was torn completely across. The intussusceptum reaches the rectum in nearly one-third of the cases—in 16 of Rafinesque's 55 cases. Even when it has protruded some inches from the anus motions may be passed from the ileo-caecal valve. The sphincter is often patulous and relaxed.

Prognosis.—The chance of *spontaneous cure* is not more than 1 or 2 per cent. This may take place either by (1) spontaneous reduction, a phenomenon several times recorded but always dubious, (2) by spontaneous elimination of the intussusceptum, or (3) by the formation of a faecal fistula above the invagination. As antiperistalsis normally occurs in the upper part of the colon, it is reasonable to believe that spontaneous reduction may occur. Spontaneous elimination of the intussusceptum with recovery is very rare. It occurred once in 189 cases at the London Hospital, and once in 68 cases at St. Bartholomew's Hospital. The fallacy of Leichtenstern's collected cases is shewn by the occurrence of spontaneous elimination in 42 per cent of his cases, 40 per cent of which died subsequently.

When treated under favourable conditions about 50 per cent of the cases recover. At the London Hospital from 1893 to 1899, 55 cases were treated with a mortality of 82 per cent, but from 1900 to 1905 the mortality fell to 57 per cent of 134 cases. Mr. F. S. Eve has published a consecutive series of 11 cases submitted to laparotomy with 43·3 per cent mortality. Mr. C. H. Fagge has published 18 consecutive cases with 50 per cent mortality. The results that may be obtained in a favourable series of cases by a skilled operator are shewn by Mr. C. Wallace's series of 20 cases with 4 deaths. Of these 2 only were irreducible, and only 4 of more than forty-eight hours' duration. The prognosis in chronic cases is much more favourable; I have operated on only 4 such cases and all recovered. Irreducibility is the fundamental point in the prognosis of intussusception in babies after treatment, for no successful case of resection has been recorded under one year old. Gibson's figures shew that on the first day 94 per cent of intussusceptions are reducible, on the second 83 per cent, and on the third 61 per cent only. When the child becomes quiet and the abdomen distended and rigid the case is wellnigh hopeless.

Treatment.—*In babies* the best results are obtained by rapid laparotomy and reduction when a surgeon is immediately available and the surroundings are favourable. Failing this, the best treatment is injection of warm water or milk, by a rectal tube and funnel with a drop of not more than 3 feet. The escape of fluid around the tube may be prevented by cotton-wool and vaseline and by holding the buttocks together. The child should be anaesthetised and the buttocks well raised. The tumour should be manipulated to assist reduction. It may be necessary to repeat the process twice or even thrice before it is successful. The child should then be placed under morphine and atropine to prevent recurrence, which is not uncommon; thus, of 23 cases treated by injection 14 required subsequent laparotomy (Eccles). *In adults*, intussuscep-

tion is seldom so urgent as in babies, and there is usually time to call in a surgeon, as the treatment is entirely surgical.

E. Volvulus, Intertwining, and Knotting.—**A volvulus** is said to be present when the intestine is twisted or has undergone axial rotation. Twisting may occur about three axes: (1) A piece of intestine may be twisted around its longitudinal axis. (2) The common form of volvulus is when a coil of intestine rotates around its mesenteric axis, but in addition there may be twisting of the intestine around its own axis at the neck of the volvulus. (3) Finally two coils of intestine possessing long mesenteries may twist or intertwine around one another, or even become knotted together; as a rule one of these two constitutes an axis around which the other twists, as honeysuckle twines around a stick. The coil which forms the axis is that which is more severely strangled. Twenty-seven cases of volvulus have occurred at the London Hospital in 13 years among 669 cases of intestinal obstruction (1 in 25).

Etiology.—The disposing causes of volvulus act in three ways: (1) By narrowing the base of attachment of a coil of intestine and so producing a pedicle; (2) by lengthening the mesentery of the coil; and (3) by leading to an adhesion at the centre of the loop; as a result the adhesion with the base of attachment forms an axis around which the loop readily rotates. The base of attachment is narrowed in many ways, adhesions may form between coils of intestine, thus producing a fixed loop; or a gland in the mesentery may become inflamed and in the process of resolution contract the mesentery of a coil of gut to a narrow pedicle. Coils of intestine which have been strangled by a hernial ring or a band may retain their constricted neck and subsequently rotate around it. Chronic inflammation in the mesentery may be set up and maintained by any persistent irritation in the bowel, such as tuberculous or stercoral ulceration. This is exceedingly common in the meso-sigmoid, as was long ago pointed out by Treitz. This chronic inflammation produces stiffness and contraction of the mesentery, which in many cases leads to narrowing of the base of attachment. An infant may be born with an unusually long mesentery to any part of the intestine, and this is especially frequent at the entero-colic junction when the caecum and ascending coil fail to adhere to the right loin. The whole of the small intestine, caecum, and ascending colon may then have a common mesentery. More often lengthening of the mesentery is acquired, as when intestine frequently descends into a hernial sac, or the coil of gut is often distended or overloaded so that it falls into the pelvis. In other cases the elongation of the mesentery appears to be due to loss of fat and relaxation of the abdominal wall, the result of repeated child-bearing or advancing years. I have collected three cases in which an adhesion of the loop of intestine formed with the base of attachment an axis around which the coil rotated. In one the centre of the sigmoid flexure was adherent to the mesentery of the small

intestine, and the flexure rotated on this axis. An inflamed coil of small intestine released from a band or hernial sac may become attached at its centre, and subsequently rotate in a similar manner around its lengthened mesentery and contracted base.

The Exciting Causes of Volvulus.—When these changes which enable a coil of intestine to undergo rotation have taken place, the final twist is produced either by some violent effort, such as lifting or straining, or by distension of the loop when, as it finds room for itself in the abdominal cavity, it may undergo rotation. In one case, under the care of Mr. Jonathan Hutchinson, jun., a volvulus of the caecum was apparently induced by the rupture of a pancreatic cyst which occurred while in the London Hospital. The volvulus was treated successfully, the cyst refilled and was drained, and the patient completely recovered. When a coil of intestine has become rotated the chief hindrance to its reduction is its distension. If this can be relieved it may even spontaneously uncoil. The twist may be prevented from passing off by the great size and weight of the coil, or by another coil being drawn across the twisted pedicle and compressing it (Leichtenstern).

Pathology.—Many degrees of twisting occur. They vary not only in extent, from half a circle to even three or four turns, but also in their tightness. When the twist is slack and the vessels are not constricted the symptoms are those of partial obstruction. The pain is colicky, flatus and even solid faeces are passed, and the distension is not very great. When, however, the twist is tight and the veins are severely obstructed the usual results of torsion of an organ result. Intense venous engorgement is produced, the bowel becomes dark purple and oedematous, and ultimately green or grey and gangrenous, the capillaries burst and blood is extravasated into the tissues. The mucous membrane pours a profuse blood-stained fluid into the twisted coil, and the serous covering exudes sanguineous serum into the peritoneal cavity. The amount of this exudation and the proportion of blood in it vary with the severity of the strangulation of vessels. The enormous distension which so rapidly occurs in a twisted coil of intestine is, however, almost entirely due to the formation of gas (*vide* p. 711). The rapidity with which it forms would appear to exclude its origin from gas-forming bacteria unrestrained by the damaged gut. Probably the gas is carbonic acid produced by the vital processes of the intestine, and is not removed owing to the venous obstruction. The consequences of this distension are well marked when the coil is large, as in the case of the sigmoid flexure. Thus, the coil of the colon may be some five inches in diameter; it is greatly increased in length, and its walls are so stretched that the longitudinal striae disappear. The peritoneum may actually split and patches of gangrene appear, but gross perforation very rarely occurs in the loop itself (Treves). Perforation when present occurs above the twist. Within a day or two, however, micro-organisms pass from the lumen of the coil to the peritoneum, and well-marked peritonitis is produced. When the sigmoid flexure is twisted the huge coil extends

up the abdominal cavity in front, forcing the small intestine backwards to the right and preventing any marked distension of the intestines above the volvulus, and the abdomen becomes extraordinarily tense. The diaphragm is forced up to the level of the fourth or even the third intercostal space, and the heart and the bases of the lungs are compressed so that circulation and respiration are seriously impeded.

Anatomy.—Volvulus is most common in the sigmoid flexure, but also occurs at the entero-colic junction and in the small intestine. Twenty-four cases of volvulus at the London Hospital were distributed as follows: sigmoid flexure 12, entero-colic 5, small intestine 7. Of Leichtenstern's 76 cases, the sigmoid flexure was affected in 45, a loop of ileum in 23, the jejunum and ileum combined in 8, but there was no case of entero-colic volvulus.

(I.) *Volvulus of the Sigmoid Flexure.*—The sigmoid flexure may (*a*) twist about its mesenteric axis, or may (*b*) intertwine with a coil of small intestine. (*a*) Twisting of the sigmoid flexure about its mesenteric axis is the common form of volvulus, and appears to be caused by chronic constipation and meso-sigmoiditis. The loaded coil of intestine falls into the pelvis and drags on its mesentery, so that this is not only lengthened but its base of attachment is narrowed. The chronic inflammation in the meso-sigmoid contracts its base and stiffens it. The flexure may rotate either forward or backward. When it rotates forward the rectum lies behind the descending colon ("rectum en arrière," Potain). This is more common. When it rotates backward the rectum lies in front ("rectum en avant," Potain). In consequence of its size the changes in the sigmoid flexure when twisted are greater in degree than in volvulus elsewhere; thus, the distension may be so great that the diaphragm is displaced to the level of the third rib. (*b*) The sigmoid flexure is intertwined with a coil of small intestine. Leichtenstern collected 21 cases, all but one in males; the ages varied from twenty-four to seventy-three. The condition is extremely rare, and no case has been recorded at the London Hospital. The loops concerned must be of considerable length. Leichtenstern states that the loop of small intestine varies from 4 to 21 inches, and the sigmoid from 12 to 40 inches. As a rule (12 in 21 cases, Leichtenstern) the coil of small intestine lies in front and forms the axis. The sigmoid pedicle is wound round it from behind forwards. The sigmoid may, however, form the axis, or the small intestine may lie behind, so that four varieties have been described. Either or both coils may undergo rotation on their own axis in addition. It is not surprising that the clinical course of this condition is exceedingly acute. Many die on the day of the attack, and nearly all within two days. The symptoms are those of ultra-acute obstruction of the small intestine.

(II.) *Volvulus of the Entero-colic Region.*—Three subvarieties of this form of volvulus have been described. (1) Twisting of the normal ascending colon about its vertical axis. (2) Twists limited to the caecum. (3) Volvulus of the termination of the ileum, caecum, and ascending colon due to the presence of a long mesentery to the ileo-colic junction. Care-

ful consideration of the cases (Curling, Fagge, Handfield Jones) regarded as examples of the first and second varieties of ileo-colic volvulus has not convinced me of the existence of these subdivisions. The ileo-colic junction often has a long mesentery, which is either a congenital or an acquired condition. It is obvious that the pendulous loop so formed may undergo simple axial rotation in either direction around its mesenteric axis, or may intertwine with a loop of the small intestine. When rotation has occurred the caecum becomes enormously distended, and may travel from the right iliac fossa to almost any part of the abdomen. Should it pass to the right hypochondrium the caecum is turned over and the ascending colon is doubled across transversely.

(III.) *Volvulus of the small intestine* may occur in two forms: (*a*) one coil may twist about its mesenteric axis, or (*b*) two loops may be intertwined. The latter is so rare an occurrence that it need not be further alluded to (see Leichtenstern). As has been already pointed out, a coil of small intestine surrounded by a band, a hernial ring, or the edges of an aperture may undergo rotation on its axis, and this indeed may be the chief cause of obstruction. It must be clearly stated that normal small intestine and mesentery cannot undergo a pathological degree of torsion, and that some congenital or acquired deformity of the mesentery or some adhesion is necessary to render volvulus possible. It is necessary to state this, for where no other cause is found for intestinal obstruction or gangrene of small intestine it is easy to assume that it was twisted. In this way gangrene from acute thrombosis of the portal vein has been regarded at the operation as due to volvulus. It is probably not unusual for more or less twisting of the small intestine to take place without any obstruction either to the lumen or the vessels, but such a twist may conduce to an obstruction from some other cause. Mr. Mayo Robson has reported two cases, which he regards as volvulus, due to the passage of large gall-stones. The ordinary form of volvulus of the small intestine illustrates what has been said of the general cases of volvulus, namely, that the mesentery is lengthened, the base of attachment narrowed, and the apex of the loop may be adherent.

A child may be born with its small intestine twisted; indeed, the distension so caused may obstruct labour. Dr. Drummond Maxwell has reported such a case, and placed the specimen in the London Hospital Museum: the volvulus implicated some 2 feet of the ileum, which was black and gangrenous. Other cases have been described by Dr. N. Pitt and Mr. Harrison Cripps.

Symptoms.—(I.) *In volvulus of the sigmoid flexure* the symptoms are in general terms those of acute intestinal obstruction, with certain characteristic signs, which make a diagnosis more or less certain and easy. The patients are nearly always men, the ratio of men to women being usually stated to be as 4 to 1; all the 12 cases of volvulus of the sigmoid flexure at the London Hospital between 1893 and 1905 were males.

The onset is, as a rule, sudden, but seldom so sudden as in strangulation by a band. The pain is severe, and exacerbations occur which are often

colicky in their nature. The characteristic feature is the early appearance of tenderness due to peritonitis. Vomiting is little marked, and in a case of my own was absent altogether. This is perhaps not surprising when we consider how low down in the intestine the obstruction is placed. Eructations, however, are common. Constipation is, as a rule, absolute, but considerable variations occur in this respect, and tenesmus is frequently present. Collapse is later in its appearance, and not nearly so well marked as in strangulation by bands. The respirations are often frequent, in consequence of the abdominal distension. The characteristic sign of volvulus of the sigmoid flexure is the rapid distension of the abdomen, which becomes extraordinarily tense, so that the diaphragm is forced upwards and the respiration and circulation are correspondingly embarrassed. The distension is so great that, as a rule, no separate coil of intestine can be distinguished; in the exceptional cases in which contracting coils of intestine are visible, the case is subacute in its onset and course. Great tenderness due to peritonitis appears early. Although volvulus of the sigmoid usually produces acute intestinal obstruction, in not a few cases the onset may be gradual and attended by colicky pains and the passage of flatus or even faeces in fair quantity. Diarrhoea may be present, and it is even probable that incomplete degrees of volvulus may either spontaneously untwist, or if they remain twisted, may only partially obstruct the bowel and its vessels. In the latter case some passing atony of the muscular coat may produce complete obstruction. On the other hand, volvulus may pursue a fulminant course, as in a man aged thirty-eight, under the care of Mr. Warren Tay in 1902 at the London Hospital; seven hours after the onset of obstruction this patient was profoundly collapsed, and the abdomen hard, resonant, and tightly distended; the sigmoid flexure was black and gangrenous, and the peritoneum contained turbid blood-stained fluid.

(II.) *Symptoms of Volvulus of the Ileo-colic Junction.*—The obstruction is usually subacute, and the onset is even less sudden than in volvulus of the sigmoid flexure. Vomiting is usually present but is not severe. The distension is not so great as in volvulus of the sigmoid, but in all the four cases of which I have notes the distended caecum was defined through the abdominal wall as a resonant tumour the size of a child's head, in two cases in the left hypochondrium, and in the other two in the right loin and iliac fossa.

(III.) *Volvulus of the small intestine* has no characteristic symptoms. It may be acute, subacute, or chronic, or attacks of obstruction may recur for a long period with spontaneous intervals of relief. In many cases the twisted coil can be felt as a tumour in the umbilical region, and vomiting is a marked and constant symptom.

Prognosis.—In the severe cases which come to operation the prognosis is exceedingly grave. In thirteen years at the London Hospital only 2 cases recovered out of 27 (mortality 93 per cent). Two further cases were successfully operated on during 1906.

Treatment consists in laparotomy: the distended coil is punctured,

emptied, brought out, and the volvulus reduced; if gangrenous it is at once resected, otherwise a Paul's tube is inserted, and, should the patient recover, the volvulus is resected subsequently, and end-to-end anastomosis performed. If the sigmoid flexure be simply emptied and reduced the volvulus is apt to recur. Blake has recorded a very remarkable case which was successfully operated on four times for volvulus of the sigmoid flexure. On two occasions the intestine was incised and emptied, and on the two other occasions a rectal tube was introduced into the volvulus after the abdomen was opened.

Torsion or volvulus of the great omentum was first described in 1882 by Oberst, who found a pedunculated portion of omentum twisted in a hernial sac. In this condition the omentum is twisted upon a more or less narrow pedicle, so that the part peripheral to the twist becomes engorged with venous blood or even gangrenous. Messrs. Corner and Pinches in 1904 collected 54 cases, 35 of which were examples of unipolar rotation, the omentum being rotated on one pedicle; this was in the upper third of the omentum in 24 cases, in the middle in 1 case, and in the lower third in 10 cases. Bipolar rotation, in which the omentum was adherent at its extremity and rotated on the axis formed by its two points of attachment, has been recorded in 6 cases. The secondary adhesion has been to a hernial sac, to the neck of a hernial sac, to the right Fallopian tube, to an appendix epiploica, or to the ascending colon.

Intertwining of pedunculated omental tumours has been reported by Heitz and Bender and by Mauclaire. In Eitel's patient, in whom the torsion was not tight and the course of the case was chronic, many gallons of ascitic fluid were drawn off by puncture.

Pathology.—Where many twists are present it is often clear that some have existed for a considerable time. These preliminary twists are probably responsible for previous attacks of strangulation which pass off as the circulation adapts itself. For torsion of the omentum to occur a preliminary moulding of this structure is necessary. In 28 out of 33 cases collected by Stewart in 1905 these preparatory changes had taken place in a hernial sac. The extremity of an omentum that habitually descends into a hernia becomes a rounded swollen mass with a neck or pedicle, and very frequently contracts one or more adhesions to the fundus of the sac or to the hernial ring. These changes in form of the omentum are the disposing causes of torsion. When these conditions are once produced the pedunculated mass of omentum may be twisted either in the hernial sac or within the abdominal cavity. The exciting causes of volvulus are: (1) Reduction and redescent of the omental mass along the inguinal canal; (2) fortuitous external movements communicated to the mass, especially by the legs in walking; (3) within the abdominal cavity unequal contraction of the abdominal wall, respiratory movements, and the peristalsis of the intestine probably convey the

rotatory impulse to an omental mass which is asymmetrically hung upon its pedicle.

Where no hernia has been present the omentum has for the most part been found adherent at its extremity. In Noble's case it was adherent to the right Fallopian tube; in Wiener's case to an appendix epiploica on the ascending colon. The omentum being attached at either end is rolled around this axis (bipolar rotation). It is, of course, evident that torsion in such a case must occur at each end in opposite directions. Scudder has recorded and figured a case in which there was no hernia, and the omentum was not adherent. The whole of the omentum may be twisted or only a part of its end or two separate pedunculated portions may be intertwined. The pedicle varies from a half to six inches in length, and may be twisted from one to eight times.

The Symptoms.—There is often a history of previous attacks, which are no doubt in many cases due to loose or reducible torsions of the omental mass. The patients are generally men of advanced years with reducible inguinal hernias of long standing, and the onset of the symptoms is frequently associated with some trouble with the hernia and forcible attempts at reduction. Where no hernia exists, or where the omentum is within the abdomen when it becomes twisted, the onset of the disease is sudden and without warning. The patient is seized with sudden and severe abdominal pain, which is general at first, but is, as a rule, later referred to the right and lower part of the abdomen. Nausea and vomiting are usually present, and are proportional to the pain. A tumour is nearly always palpable unless the abdominal wall be too rigid or fat, and frequently is in the hernial sac or just inside the hernial ring. In other cases it is in the iliac fossa, and extends a variable distance up into the abdomen. The tumour increases in size, it is ill-defined, tender, immovable, and dull on percussion. After a day or two the signs of free fluid appear in the abdomen, and should an empty hernial sac be present this is full, soft, and fluctuating. The abdomen becomes distended, and is rigid, especially over the tumour. The temperature is, as a rule, slightly raised owing to the extravasated blood. The pulse is seldom over 100 per minute, unless the haemorrhage be excessive. The bowels are constipated, but, as a rule, flatus is passed, and enemas obtain a tardy result.

Diagnosis must be made from incarcerated hernia, reduction of a hernia *en masse*, appendicitis, a twisted ovarian tumour, or intestinal obstruction as in Audier's case. Of the first 29 cases, collected by Rudolf, 1 only had been diagnosed correctly.

Treatment is surgical. A hernia if present is explored. An incision is then made on the tumour, and when the infiltrated omentum has been freed from adhesions, ligatures are applied above the twisted pedicle, and the mass removed. In Eitel's case with chronic ascites the omentum was unrolled and spread over the intestines; the patient was cured.

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DISEASES OF THE COLON

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I. Simple Colitis.—By this is meant a catarrhal inflammation of the large intestine, comparable to gastritis or to bronchitis. For example, a patient was examined with a speculum, and as far up as we could see, and in all probability much farther, the mucous membrane was intensely injected, of a bright red colour, swollen, and secreting a thick mucus ; but no ulcer was visible. The patient quickly recovered under appropriate treatment. We have post-mortem evidence of a more severe condition. Wilks and Moxon mention a "case attended by discharge of mucus and blood in which, after death, the whole internal surface of the colon presented a highly vascular, soft, red surface covered with tenacious mucus or adherent lymph, and here and there shewing a few minute points of ulceration. The coats also were much swollen by exudation into the mucous and submucous tissues." A very well-marked instance was admitted into Guy's Hospital under Sir Cooper Perry on April 19, 1891. A woman aged 36, in her seventh month of pregnancy, was slightly sick for the fortnight previous to April 13. At the latter date the vomiting became worse. Her face was sunken, the rectum was empty, the bowels were constipated, but for the last few hours of life she had diarrhoea. She died soon after admission. The whole colon was inflamed, the mucous membrane being thick, sodden, and deep purple ; no ulceration and no membranous exudation could be seen. The liver was fatty.

There are two varieties of simple colitis ; one, which is not associated with any other serious malady, may be called *acute primary colitis*, to distinguish it from the results of direct injury or extension from neighbouring parts, and from colitis associated with constitutional diseases, such as Bright's disease, or with local disease, such as cancer, of the bowel. Acute primary colitis, said to be commonest in children, is, no doubt, always due to a micro-organism. Sometimes it occurs as an epidemic.

Dr. A. E. Carver has recorded a severe outbreak with a high mortality; of his cases many were children. The motions were often solely composed of blood and mucus. At the post-mortem the large intestine was oedematous, its interior was covered with mucus, it was reddened, there were submucous haemorrhages, and in many places the process had gone on to ulceration. The *Bacillus enteritidis sporogenes* was constantly present. Dr. Cautley has described a similar epidemic, and has pointed out that these cases may go on to chronic colitis. Acute colitis has been called mucous colitis; but as this name has been also used to describe what I shall call membranous colitis, it is liable to lead to confusion. Of several cases under my care, the three following are good examples of acute primary colitis:—A lady aged 25 was suddenly seized with pain in the abdomen, quickly followed by diarrhoea, which became very frequent; the motions consisted chiefly of mucus, but they contained some bright blood. Pressure over the transverse and ascending colon caused some pain. The tongue was covered with a very white fur. She had loss of appetite and nausea, but no vomiting. The temperature varied between 99° and 101° F. The second case, a man aged 30 years, was suddenly one morning seized with oft-repeated severe diarrhoea. The motions at first consisted of nothing but mucus and blood. In the course of five days the diarrhoea slowly ceased and the motions contained more faecal matter. His temperature gradually mounted, till on the fifth day it was 104·2° F. Then there followed marked constipation. On some occasions he had much severe griping pain, chiefly along the course of the colon, when the bowels were open. There was also abdominal tenderness. He recovered completely. The third patient, a man aged 38, was suddenly seized with diarrhoea; the motions contained much mucus and blood, and the microscope shewed the presence of red blood-cells, leucocytes, and triple phosphate crystals. He had considerable griping pain, abdominal tenderness along the colon, and loss of appetite. His tongue was covered with a white fur. For three months this train of symptoms continued and he lost 20 pounds in weight. He then consented to take to his bed, and from that time he began to improve.

Symptoms.—The main symptom of this disease is a diarrhoea, which may come on suddenly; there is much mucus in the stools, and often blood also, even in considerable quantities. At first there may be absolutely no faecal matter; but as the patient improves the motions contain more faeces and less mucus. The blood is mostly fluid, and but little changed either to the naked eye or the microscope; so we infer that it has but recently left the vessels, and has come, therefore, from the large intestine. Often the mucus is in little lumps. Triple phosphate crystals may be seen on microscopical examination; and, more rarely, oxalate of lime, cholesterin, and Charcot's crystals. In exceptional cases the motions have been described as frothy. The bowels may be opened many times a day; tenesmus is often present, but it is not by any means always a striking feature. A digital rectal examination reveals nothing abnormal, except that sometimes the mucous

membrane feels a little rough, but sometimes the inflammation may extend low enough to be seen with a speculum or sigmoidoscope. Some patients pass much wind from the anus, and complain of rumbling or of borborygmi. Abdominal pain is a common symptom; it comes on in paroxysms, often associated with defecation, but it bears no relation to food. It is of a griping character, often very severe: and it nearly always follows the course of the colon. Between the attacks the patient may be free, but he sometimes still complains of a dull pain. Abdominal tenderness is usually present; a common seat for it is over the sigmoid flexure, but the whole colon, or even the whole abdomen, may be tender. According to Habershon, some of these patients suffer from irritability of the bladder and dysmenorrhoea. In acute colitis, as in other diseases of the large intestine, the disease may closely resemble appendicitis if the appendix be affected, and it is important to bear this in mind, for excision of the appendix fails to cure the disease. Dr. H. A. Caley has especially drawn attention to these cases.

There may be pyrexia; and the temperature may remain raised even when the patient is much collapsed from diarrhoea and loss of blood. The pulse is rapid, and, in a severe case, small, soft, and running. The abdomen usually moves with respiration, and is not commonly much distended. Some patients vomit, there may be considerable nausea and loss of appetite, thirst may be a cause of complaint, and if the diarrhoea be unchecked, anaemia and loss of flesh may follow. The tongue is furred, and it is important to remember that the fur is almost pure white, whereas in many diseases with which colitis may be confounded it is a brownish- or yellowish-white. The fur is often universal, but the tip and edges may be clean. As a rule these patients suffer much from mental depression; they exaggerate trifles and take a gloomy view of life. In some patients the neurotic element is so marked that it forms the chief feature of the case; we are forcibly reminded of the neurotic dyspeptic invalid. In my experience this neurotic variety is more common in men than in women. Such, when they come to see the doctor, frequently bring with them a written statement containing a minute account of all the symptoms, and often a careful description of the motions, and are valetudinarian in aspect and behaviour. These cases are often very chronic, and they are most difficult to cure.

Various amoebae, infusoria, and other micro-organisms have been described in colitis, and may perhaps be the cause of the disease. When the inflammation is limited to the sigmoid it has been dignified with the name sigmoiditis, and no doubt the long retention of faecal matter here and in the rectum leads to some inflammation, but any symptoms due to such sigmoiditis or proctitis are usually overshadowed by those of the constipation (see Pericolitis, p. 1015).

The reader must not suppose that simple colitis is always so serious as the above description might lead us to suspect; but the difference is only one of degree. The majority of cases are mild, when the symptoms are all less intense; and probably many cases of slight

diarrhoea, which it is difficult to classify, are really instances of mild colitis. On the other hand, the diarrhoea may be uncontrollable, and the patient may die from exhaustion. I once saw a man who, when convalescent from a severe and prolonged attack, died with all the symptoms of pulmonary embolism.

Diagnosis.—This, as a rule, is not difficult. The disease is known from ulcerative colitis by its sudden onset, by the large amount of mucus in the motions, by the early appearance of blood in the case, and also because under appropriate treatment it generally yields. In England acute dysentery is hardly likely to cause much confusion; but the "meat washing" character of the stools, the burning pain in the rectum, the intense tenesmus, and the constant desire to go to stool even when nothing is passed, should prevent a mistake. Nevertheless, we often see severe cases of diarrhoea in persons who have had dysentery abroad; and I think that those who have had dysentery are particularly liable to colitis. I have seen some extremely bad cases, but under very strict treatment for the colitis the patients all recovered. The character of the motions and the sudden onset distinguish colitis from enteric. It should never be forgotten that malignant disease of the bowels often presents itself in such a guise as to lead to errors of diagnosis. I have known a case diagnosed as colitis turn out to be chronic arsenical poisoning.

The *prognosis* is, as a rule, good. Recent acute cases, except in the rare instances in which they are fatal, recover quickly; but in cases allowed to become chronic—and often the disease has lasted many months before it is seriously taken in hand—the patients require rest in bed and milk diet for many weeks.

The *treatment* should be on the same general principles as that of diarrhoea (p. 668). The patient must be kept absolutely in bed until the diarrhoea has stopped, the motions are well formed, blood is no longer passed, and the temperature is normal; and longer still if he has had a severe attack. Warmth to the abdomen is very desirable. He should consume nothing but milk, and should not take more than two fluid ounces at a time. The total daily amount will depend upon the acuteness of the attack and the general condition of the patient; usually he can take from two to three pints a day. A good way of checking the diarrhoea is to give some laudanum with fifteen or twenty grains of carbonate of bismuth suspended in some mucilage every four hours; the compound kino powder is also very valuable: if these drugs fail, a starch and opium enema will often succeed. Should opium for any reason be contra-indicated compound catechu powder is very useful. I have tried washing out the large bowel with a tepid saturated solution of boracic acid, but I do not think there is much advantage in this. All astringent drugs should be omitted as soon as possible, for the constipation that follows colitis is usually very troublesome. It is best relieved by a rectal injection of six or eight ounces of warm olive oil or a drachm of glycerin. A long holiday among new and interesting surroundings greatly aids the convalescence in the neurotic cases.

Secondary colitis, by which I mean that which occurs either by extension from neighbouring parts or in association with some grave morbid condition, is occasionally seen. Among the records of autopsies at Guy's Hospital I find the following varieties :—

i. *Colitis due to Direct Extension.*—I made a post-mortem examination on a woman who died from swallowing sulphuric acid. All along the greater curvature the whole thickness of the stomach was gangrenous, and the transverse colon was red and black on its exterior, and acutely inflamed on its inner surface. The stomach was in contact with the colon. The small intestine was unaffected, so it appeared that the effect of the acid had spread through the walls of the stomach to the transverse colon (59). Proctitis may be gonorrhoeal, and then it usually spreads directly from the vagina.

ii. *Colitis due to Direct Irritation.*—There is in the museum at Guy's Hospital the colon of a man who died in 1827 with severe diarrhoea, induced by large doses of purgatives given to overcome severe constipation. The colon is black, gangrenous, and sloughy. The colon may also be inflamed as the result of corrosive sublimate or arsenic given by the mouth, even when the small intestine is unaffected ; probably because the poison, after having been absorbed higher up, is excreted into the colon. The proctitis due to *Oxyuris* and *Bilharzia* (*vide* Vol. II. Part II. p. 875), and that due to direct injury, are examples in point. The varieties of proctitis are not described in any detail here, as they are usually considered in textbooks of surgery.

iii. There is a form of *acute colitis associated with septic or pyaemic conditions.* Of the five following examples, the first three are instances of an early stage of the process. A woman, aged 40, was admitted under Mr. Davies-Colley in 1883 with chronic cystitis and suppurative pyelitis. "Rather intense colitis, the mucous membrane being rough, granular, ecchymosed, and swollen," was found. In 1886 a girl was admitted for an extensive burn. Three days before death she was seized with vomiting and diarrhoea. There was pus in the right knee, the colon was vascular in patches, the solitary glands were enlarged, and Dr. Goodhart thought the condition one of early inflammation. In the same year a girl died from cancrum oris. The colon was injected, thickened, and covered with flakes of lymph. Also in 1886 a boy was admitted for compound comminuted fracture of the right tibia and fibula. Pyaemia, with pus in several joints, appeared, and he died a fortnight after admission. The mucous membrane of the rectum was rough, thickened, and vascular, with numerous minute ulcers and patches of yellow lymph. Higher up in the colon there were similar but less severe patches. There was a large patch in the caecum and many sloughs in the colon. Dr. Goodhart remarks that the kidneys were exactly like some I described in the 26th vol. of the *Trans. Path. Soc.* ; that is to say, scattered throughout the organs were numerous patches consisting of an outer red zone, then a yellowish zone, and most centrally a brown area. A still more extreme colitis occurred in the case of a

woman aged 28, who was admitted under Dr. Galabin in 1888 with pelvic abscesses and peritonitis from gonorrhoea. In the caecum and for two feet above it the mucous membrane was sloughing; and from beyond this to the anus the colon was acutely inflamed, its walls were oedematous and thickened, and its mucous surface was greyish-yellow with patches of submucous haemorrhage. The mucous membrane was separating in shreds in many places. It will be noticed that three cases had no remote pyaemic lesions. In no case is it possible to fix the duration of the colitis. It is noteworthy that, although this is a rare condition, three cases occurred at Guy's in 1886.

iv. *Acute colitis* sometimes occurs *in association with Bright's disease.* A man, about 59, had chronic Bright's disease; he passed large quantities of mucus; the surface of the colon down to the rectum was covered with mucus and catarrhal products. Another case was that of a man, aged 31, who had gout, granular kidneys, and mitral stenosis; there was much bloody diarrhoea. After death, at the lower part of the descending colon there was injection of the mucous membrane and a small quite superficial ulcer; near the rectum was another patch of injection and another small ulcer. The Guy's Hospital Museum contains three specimens of colitis in Bright's disease. The first was from a man, aged 32, who had large white kidneys. The colon happened to be sacculated; it was thickened, and had many sloughs on its mucous membrane; they were for the most part on the prominences between the sacculi. The second was from a man, aged 45, who also had tubal nephritis; the colon was inflamed, sloughing, and in parts black and gangrenous. The third was from a man, aged 25, who had tubal and interstitial changes in his kidneys. Here also sloughs were present in the colon, and there was superficial ulceration. Sometimes the colitis of Bright's disease is associated with acute inflammation of the small intestine, as in an instance recorded by Dr. Goodhart (33).

Perhaps some of these cases should be regarded as an early stage of ulcerative colitis, which, as I shall presently shew, may be associated with chronic Bright's disease. Acute colitis is, I think, not so rare a complication of Bright's disease as is commonly supposed, but as the patients are usually extremely ill from nephritis very little can be said of the symptoms. In all the cases there was severe diarrhoea; this, however, is a symptom of uraemia, and moreover purgatives are ordered for many cases of Bright's disease. Very little stress can be laid upon haemorrhage, as persons with Bright's disease may bleed anywhere.

v. *Colitis is associated with pneumonia* in extremely rare cases, but I have seen pneumonia ushered in with diarrhoea probably due to pneumococcal colitis. Two specimens are to be found in our museum: one from a man, aged 20, admitted for pneumonia: his febrile symptoms lessened, but severe diarrhoea coming on, he sank into a typhoid condition and died. The whole of the large intestine was acutely inflamed, and shewed a few small rounded ulcers. In the second case, on the eleventh day of the illness, diarrhoea set in, and six days later the patient died.

The colitis had proceeded to the stage of the formation of shallow ulcers. That the pneumococcus can affect many parts of the body is well known. Perhaps, therefore, it may directly attack the colon. It is possible, however, that some of the specimens of colitis found in the bodies of persons dead of septicaemia, Bright's disease, or pneumonia, may represent early stages of the membranous colitis which may complicate these diseases. Acute colitis has been known to be associated with tuberculosis and actinomycosis, and with an abdominal aneurysm.

vi. If *the colon is lardaceous* it may, like the rest of the lardaceous bowel, become, in very exceptional instances, acutely inflamed.

II. **Membranous Colitis.**—Disease of the colon leading to the production of a membrane is not very common. Two distinct varieties exist: in one, called dyspeptic membranous colitis, the membrane is always composed of mucus, there is no demonstrable inflammation of the colon and other disease is rarely present; in the second, which is always associated with some other grave condition, or is due to direct injury, and might therefore be called secondary membranous colitis, there is demonstrable inflammation and the membrane is not composed solely of mucus. It is not usual to include under the heading of membranous colitis those cases of constipation in which when the bowels are open shreds of coherent mucus are passed with or without the hard masses of faecal matter. Such cases are common enough.

Dyspeptic membranous colitis is so called because the sufferers from it complain much of dyspepsia, and they pass membranes from the anus. Many other names have been used; a list of them will be found in an article by Dr. Light, who terms the condition desquamative enteritis; but it seems to me that this phrase fails to express that distinct membranes are passed, and that the chief seat of the disease is probably the colon. Many references are given by de Langenhagen, who also enumerates the several names which have been applied to the malady; such are mucous colic, glairy enteritis, glutinous diarrhoea, mucous affection of the intestine, and intestinal croup. Perhaps the most accurate is muco-membranous entero-colitis, but inasmuch as the membranes are always composed of mucus and the disease is usually limited to the colon, this cumbersome name may be, and usually is, shortened to membranous colitis. But in using it we must remember that the membranes are composed of mucus and are quite different from those passed in secondary membranous colitis, and that there is in this disease no demonstrable inflammation of the colon.

The disease usually begins between the ages of 20 and 40; it is rare over 50, but I have seen two cases over 70; it is commoner in women than in men in the proportion of 5 to 1, and in private than in hospital practice. It is rare, but no doubt is often overlooked. In children it is excessively rare, for Edwards found that out of 111 cases only six were under the age of 10; of my 60 cases one only was under 10. The distinguishing feature of it is that membranes, which the patient usually calls "skins," are passed from the anus. One patient

thought he was passing pieces of tapeworm; and this gives a good notion of the common consistence and colour of the membrane, which, however, may be brown from faecal staining. In an extreme case it forms a complete tubular cast of the intestine, usually from one to six inches long; but such tubes have been known to be several feet in length, and sufficiently thick and tenacious to admit of their being held up. The wall is occasionally laminated, and pieces of faeces may be detected between the laminae, shewing that they have been laid down successively. Faecal matter may also be found in the interior of the tubes, the diameter of which may be anything up to 1½ inch; and the thickness of wall varies from an extreme tenuity to one-fourth of an inch; the end may be well defined, but often it shades off into a soft, transparent, gelatinous material. The tubes may be marked, apparently, by the valvulae conniventes, or they may have a sacculated appearance, indicating their origin in the colon; their outer surface is smooth, the inner is more rough, probably from the attrition of the faeces. Either with or without the tubes shreds of all shapes and sizes may be passed, but in all essential characteristics they are the same as the tubes. Often several pieces of membrane, when passed, are rolled up into a solid ball. Under the microscope the membranes are transparent and structureless; embedded in them may be seen minute fragments of undigested food and faeces, some cells, free nuclei, putrefactive micro-organisms, phosphates, and cholesterin crystals. The cells are apparently the epithelial cells of the large intestine which have undergone fatty degeneration. Groups of them may be seen compressed together in rows. When the inner surface of the membrane is magnified it appears to be reticulate, and presents at regular intervals depressions or even perforations which clearly correspond to the mouths of Lieberkühn's crypts. Often the cells previously described are grouped around these openings in such a way as to shew that the lining of a follicle has been cast off and become incorporated in the membrane. In Dr. Goodhart's case the pits were much larger than healthy follicles, from which it would appear that the follicles had been distended. Chemically the membrane consists of mucus.

The reader who wishes to picture to himself a patient suffering from this disease should have in his mind a chronic dyspeptic woman of a depressed turn of mind and suffering from constipation; though the malady may also occur in the busy, energetic, nervous subject. The *symptoms* of the chronic dyspepsia are that the patients are poor eaters, believing that first this and then that article of food disagrees with them, so that their diet soon becomes very limited in choice and quantity; they are usually anaemic, thin, and complain of the cold; the tongue is pale and covered with a very white fur; and the bowels are nearly always constipated, often very severely; but the constipation is occasionally alternated with mild diarrhoea. In rather rare cases there is no constipation but only diarrhoea, and the patient then usually traces the onset of the disease to an attack of diarrhoea. Some patients suffer from an irritable bladder, and may pass much mucus in the urine.

Da Costa mentions a sense of rawness and even acute tenderness in the abdomen as occasional symptoms, and he states also that sufferers from this disease are very liable to boils. They are often taciturn, rarely have buoyant spirits, and not infrequently suffer from great depression, taking a gloomy view of life and exaggerating the importance of trifles. These dyspeptic symptoms and this frame of mind are more or less constantly with the patient, but there are exacerbations from time to time, during which there is much abdominal pain, usually griping, generally somewhere in the course of the colon, and often coming on a long while after food; the abdomen is tender, especially over the colon; flatulence is troublesome, the loss of appetite and constipation are still more marked; the patient may complain of nausea, or suffer from vomiting; the tongue is more furred, and may be rather red; and the mental depression and feeling of general weariness are very pronounced. After some time the patient notices that the "skins" are passed by the bowel. In a mild case this lasts for a few days only, during which the dyspeptic symptoms may be less marked; she is then restored to her usual feeble health till another attack comes on; but in more severe cases the passage of membranes and the other symptoms continue for weeks or months, and much bright blood, which has clearly come from low down in the bowel, may be seen. In fact, sometimes when the bowels are open nothing but blood and membranes are passed. Two fatal cases I have seen seem to shew that the membranes are passed soon after they are formed, for hardly any were found at the autopsy. The membranes in the motion are often compressed into a tight ball about the size of a nutmeg; and when faecal matter is passed it is in the form of a few small, hard, scybalous masses with pieces of membrane between them; even if ordinary faeces do come away, the first part of the motion usually consists of membranes only, or of the thick, transparent, tenacious, jelly-like material that occasionally takes their place: in Dr. Shingleton Smith's case, however, the faeces sometimes preceded the membranes. While passing them the patient is, for the most part, unusually constipated, and often takes enormous quantities of purgatives, but occasionally the bowels may be opened two or three times a day. The act of defecation is often excessively painful. I have known a patient sit on the water-closet for a couple of hours suffering from agonising griping pain before a motion consisting of enough blood and mucus to fill a half-pint measure was passed. She often used a morphia subcutaneous injection immediately before defecation, and twice she fainted in the water-closet from the severity of the pain. Occasionally the faeces are very pale. A rectal examination reveals nothing abnormal. The abdominal griping pain is usually less after the bowels have been opened, and for a few hours the patient may feel a trifle better; but otherwise, unless the case be mild, the general symptoms present no such remissions, and a dull constant pain is often present, but in rare instances there is no pain of any kind. Dr. Goodhart's patient complained of feeling cold, and sometimes had rigors when the bowels were relieved. It is common when the hand is placed on

the abdomen to find that the sigmoid and even the descending colon feel thickened and can be rolled about under the hand like a sausage. This is not due to any actual thickening but because it is tightly contracted. One patient passed a moderate amount of uric-acid gravel. Dr. Light says that an excess of urates may be present, and that the subjects of this disease are liable to urticaria. With these exceptions the functions of the body are usually normal. The cases vary very much in severity. One patient I see occasionally has an attack, lasting about a week, on the average once a year. In the intervals she has fair health. On the other hand, many patients pass these membranes for months, or even for many years; all the while they are perfectly wretched from chronic dyspepsia, they waste, become extremely anaemic, live in constant dread of defecation, and either lie constantly in bed too ill to move, or spend all their money in travelling from health resort to health resort.

Certain associated conditions call for notice, and my series of 60 cases (85) shews that these are particularly frequent in patients over 50. Membranes composed of mucus may be passed by those who have organic disease of the bowels; thus, I have seen them passed by those who have had malignant disease of the gut, by those who have had appendicitis, and by those who have had other inflammatory masses in the neighbourhood of the intestines. These associations are of great importance, for if they are forgotten the patient may be considered to be suffering from membranous colitis only, when in reality serious organic disease is also present. Not only may an ordinary severe attack of appendicitis produce membranous colitis, but if the production of membrane take place throughout the whole of the large bowel, the formation of it in the appendix and caecum gives rise to pain and tenderness there, and the patient is erroneously thought to have ordinary appendicitis. In such a case removal of the appendix does not lead to a cessation of the formation of membrane or to the relief of any of the symptoms, but if the membranous colitis be induced by attacks of ordinary appendicitis, removal of the appendix will effect a cure. Over 40 per cent of the women afflicted with membranous colitis have trouble with the organs of generation; the associated diseases in these organs are most varied: pelvic cellulitis, endometritis, and dysmenorrhoea are all common, but the most interesting is membranous dysmenorrhoea. This is met with in about 10 per cent of all cases of membranous colitis, and it is strange that the passage of a membrane composed of mucus should be associated with the passage of shreds of mucous membrane from the uterus. In one case the urine contained a great deal of mucus. General visceroptosis and a floating right kidney are both more often seen in those women who have membranous colitis than in women generally. Sometimes the various associated neuroses are so striking as almost to amount to complications; the symptoms of hysteria, neurasthenia, and hypochondriasis are very common. One patient I saw became insane and died.

The passage of *intestinal sand* is an interesting complication of

membranous colitis. The exact proportion of cases in which it is passed is not known, and often no trouble is taken to look for it, but it cannot be less than 10 per cent. This sand, which is very fine, is seen in the bed-pan with the motion, from which it may be separated by washing. Usually it is red looking and somewhat like fine uric-acid gravel, but it may be dirty white. The amount of organic matter in the sand varies from 30 to 70 per cent; the inorganic matter is almost all calcium phosphate with traces of calcium oxalate, magnesium, iron, and perhaps silica. The colouring-matter is usually urobilin. The amount of sand passed in a day may be four teaspoonfuls, but commonly it is much less. Many patients pass it for years, but even then not always constantly; it may be passed daily for weeks, and then for weeks none may be passed. Although milk contains much calcium, this sand is probably not derived from the food, for milk diets are so common and intestinal sand is so rare. It is known that the mucous membrane of the intestine secretes lime salts, but why such large quantities of calcium phosphate sand should be passed in membranous colitis is not clear. Those who pass it have a severe degree of the disease; it is seen much more commonly in this than in any other disorder, but it may occur in other chronic diseases of the large intestine. It must be carefully distinguished from false intestinal sand, which looks very like it and is met with in those who have partaken largely of pears. The vegetable nature of this is easily recognised under the microscope.

A few of the cases have piles, and when the piles are operated upon no benefit to the membranous colitis follows; nor is this surprising, for both the piles and the membranous colitis are probably due to the same cause, namely, constipation.

While it is undoubtedly true that there may be an excess of mucus secreted by the intestine when it is inflamed, yet judging from what I have seen at post-mortems, from what I have seen in patients who have been operated upon, and from what I have read in published descriptions, there is, I believe, no doubt that Nothnagel is correct in saying, "The condition may exist without any structural evidence of enteritis; in that case it is really not an inflammatory process but one that may be termed simply colica mucosa, or, as Ewald aptly suggests, myxoneurosis." Nothnagel, like many others, regards the disease as primarily a nervous disorder with an excessive secretion of mucus from the colon as one of its symptoms. The chief arguments in favour of this view are that the disease is especially common in women and is frequently associated with severe and various nervous symptoms, *e.g.* it and asthma have existed in the same patient, and hysteria and various neuroses are common associations, but it must be remembered that it is often associated with diseases of the female pelvic organs, *e.g.* pelvic cellulitis, which can hardly have a nervous origin. But there are some strong reasons for regarding the malady as a local disorder of the colon: first, it may undoubtedly be associated with definite organic disease of the bowel, *e.g.* cancer and appendicitis, and when we bear in mind the close anatomical association

between the upper part of the rectum and the rest of the pelvic organs it may be that in some cases at least in which these are diseased the membranous colitis is directly due to such disease. Secondly, this form of colitis is almost always associated with constipation, and in a few cases it appears directly to follow some diarrhoeal disorder. An argument against the view that constipation is the cause is that, although constipation is so frequent, membranous colitis is not common; but a very strong reason for regarding the constipation as causative is that all successful modes of treatment are local and have for their object keeping the large bowel empty. Thirdly—but this is not a strong reason—in some cases there are two abnormal secretory results, viz. an excess of mucus and much calcium phosphate, and perhaps it is a little more likely that these would both follow a local cause than a functional disease of the nervous system. It would be much against regarding the disease as local if it could be shewn that when it follows local disease no nervous symptoms are present, but this has not been done, and certainly could not be done in all cases. Probably, therefore, the disease is primarily a local disorder of the large intestine. In this opinion I am supported by Tuttle.

Prognosis.—The disease is hardly ever fatal. In not one of my 60 cases did the disease itself kill, but about a third of the cases were not benefited by treatment; rather over a half were cured, and the remainder were much improved; other authors record more favourable results, but my cases were all sufficiently ill for a second opinion to be sought. By far the most important point in estimating the prognosis is the duration of the disease. When, as often happens, the patient has suffered for years before proper treatment is begun, recovery is rare; on the other hand, if the patient has not been ill long she will probably get better. Men do better than women. Age does not affect the prognosis. Other things being equal, severe cases are more difficult to cure than mild. Sometimes many months of treatment are necessary before improvement begins. If the patient die, it is from some associated condition.

Treatment.—Between the attacks the patient should in all respects lead as healthy a life as possible. She should take plenty of outdoor exercise of a kind to interest her and take her out of herself; riding or playing golf is infinitely preferable to dull, solitary walks taken merely for the sake of the exercise. The diet should be ample; most of these patients decline first one article of food and then another, until at last their dietary is so restricted that they are not only underfed, but their digestive powers are overtaxed in some particular direction. An obviously indigestible dietary, such as one consisting largely of rich dishes, or an undue preponderance of sweets, should be avoided; but the patient should partake of any ordinary food that is put before her. It should be well cooked, and the meals should be made as tempting as possible to the slender appetite. Even if it be an effort, she must eat well. She should have her meals at regular times, and should go to bed early, and always have eight hours' sleep.

Some have advised that the food should be predigested, and others, as von Noorden, have advised that the diet should contain much bulky indigestible food. I have known both these succeed, but ordinary diet succeeds as often as either, and has the advantage that it does not foster valetudinarian habits. As these patients are always worse if they are idle, they should have some occupation that will constantly employ the mind. Certainly in some cases the daily passage of high-frequency currents through the abdomen seems to help. A holiday and change of scene to some such bracing place as Switzerland or Norway, or a yachting cruise, are often of the greatest benefit. Indeed, these patients derive much benefit from such a holiday, even when they are not passing membranes. In every case the attempt should be made to keep the large bowel empty. Patients rarely improve unless this is done. The simplest means is to give castor oil. The patient should take it every morning on waking, and if, as many people do, she wakes about five A.M. and falls asleep again, she should take it about five; the dose should be such as to ensure that the bowels are open comfortably and thoroughly after breakfast. It does not matter how it is taken, but often half an ounce or an ounce is required. This is certainly by far the best drug for this disease, and many have recovered as a result of the use of it. Therefore even if at first it is disliked, efforts should be made to overcome the distaste for it. If this is impossible, sulphate of magnesium may be employed instead, and if this too disagree, calomel overnight should be tried; but whichever drug be finally chosen it should be given for many months—indeed, some patients find they keep in best health if they take castor oil every morning constantly. A very considerable proportion of those afflicted with this disease recover if they take castor oil, but should it or other aperients fail then the large bowel should be washed out daily for a few weeks. If this is done in England, plain water of a temperature of 100° F. is best, between one and two pints being used. Medicated waters often do harm, and lavage in this country is never so successful as it is when carried out at Plombières—indeed, if simple aperients fail the patient should make every effort to go to Plombières, but the season only lasts from May to September.

For a severe case, rest in bed, morphine injected subcutaneously, and the application of hot fomentations to the abdomen, may be necessary; but opiates should not be prescribed unless they are absolutely necessary; for not only do they increase the constipation, but sufferers from membranous colitis are just the sort of people who may become addicted to an excessive use of these agents.

When the patient has tried all other means of relief without success, and when suffering is so great that life is a burden, the colon may be opened on the right side, to allow the faeces to pass through the artificial anus for some months, and thus to give the diseased bowel complete rest. The artificial anus may then be closed. The first published case in which this treatment was adopted was brought before the Clinical Society in 1895 by Mr. Golding-Bird and myself. The patient was

much benefited by the operation, but unfortunately she died (probably from the bursting of a pelvic abscess connected with old-standing pelvic trouble) five weeks after closure of the wound. Dr. Simpson has published a case in which colotomy was done for membranous colitis in 1894 by Mr. Keith, and to him I believe belongs the credit of having been the first to perform the operation. The artificial anus was not closed for seven and a half months. Two years after, the patient was very well; only once or twice had she passed any muco-membranous matter. In February 1896 I saw a lady who had been unsuccessfully treated for five years, and whose life was a perfect burden to her. Right colotomy was performed, the wound was left open nearly a year, and the patient improved immensely in all respects for a time, but about two years later the membranous colitis returned. Mr. Golding-Bird and I had another case which we heard of from time to time for years after the operation, and her colitis did not return after the wound was closed. Our experience is that the artificial anus should be left open for at least a year. There are serious objections to this operation; in the first place a person with a right-sided colotomy may find herself in a very disgusting condition, for the faeces passed are so fluid that she cannot be easily kept clean, and the irritation of the faecal matter may make the skin very sore; secondly, after the wound is closed the disease may relapse; thirdly, it is quite possible that when the disease has lasted many years the patient has become so accustomed to her neuroses that, even when cured of her membranous colitis, the neuroses may remain as they may after stitching a kidney. The result is that most patients decline operation, for, as the disease is not fatal, they prefer the ills they have to those which are unknown. Should this operation be done, there is no need to wash through from the artificial anus to the natural anus, and the opening must of course be right sided. Gibson gets over the disagreeable effects of a right-sided colotomy by making a valvular opening in the bowel. It has been suggested that shutting off the large bowel by short-circuiting from the lower part of the ileum to the rectum would be a better mode of treatment; it is certainly in every respect a better operation: the only objection to it is that the rectum itself is often diseased, and in a severe case it would be difficult to implant the ileum below the disease, but probably it is on the whole to be preferred to colotomy. Some operators have opened the end of the appendix, fastened it to the abdominal wound, and then irrigated from the opening to the anus, and so have kept the large bowel empty.

Secondary Membranous Colitis.—This seldom gives rise to any symptoms. Patients affected with it hardly ever pass membranes by stool.

i. Injury to the colon may be the cause, as in the case of a little boy admitted under Sir H. Howse in 1891. A cab-wheel passed over his abdomen and he died in three days. The mucous membrane of the colon was found deeply injected, and had a thin layer of granular lymph on it; and the muscular and serous coats were lacerated in several places. More often the injury is done by some corrosive substance, as in the case of a

woman who swallowed three ounces of dilute sulphuric acid. It was found that a large portion of the mucous membrane of the stomach was detached, and the colon and the lower part of the ileum were covered with false membrane. A woman who was in Guy's Hospital, nearly fifty years ago, for a strangulated hernia, had taken much purgative medicine and had swallowed a quantity of duck-shot. The hernia was easily reduced, but she died, and the whole of the colon and the last three feet of the ileum were in a state of membranous inflammation. Another woman swallowed a quantity of corrosive sublimate, and the whole colon was found in a condition of membranous colitis. The mercury was probably absorbed high up in the alimentary tract and excreted into the colon, which is certainly one channel of excretion for this metal. The possibility of this is shewn by the cases which Virchow brought before the Berlin Medical Society, in which severe membranous colitis was present as a result of mercurial poisoning following inunction and intravaginal injections of corrosive sublimate. Liebreich mentioned that he had produced the same condition in animals by subcutaneous injection.

ii. The pyaemic variety of secondary membranous colitis is so neglected in textbooks that I will briefly quote some cases from the post-mortem records at Guy's Hospital. 1893.—(*a*) A woman, aet. 68, had suffered three or four days from a strangulated umbilical hernia of the transverse colon. In the caecum and ascending colon there was extensive membranous colitis; where the mucous membrane was not covered with a thick white pellicle it was reddened. (*b*) A woman, aged 30, was prematurely confined on July 24. She had severe fever and abdominal pains, and died August 2. With the exception of the lower part of the descending colon and the sigmoid, the large intestine from the anus to the caecum was covered with a greenish-black membrane. The walls were thick and sodden. The kidneys weighed 17 ounces, and were in a state of acute non-suppurative inflammation. 1892.—(*c*) In a woman, aged 38, the following events took place: premature labour, craniotomy, version, subsequent pyaemia, death in ten days. More than fifteen inches of colon near the sigmoid were covered with disseminated patches of membranous inflammation distributed upon healthy mucous membrane. The membrane was dark. General peritonitis and abscesses in the liver and kidneys were also present. (*d*) A man, aged 71, had gangrene of the foot; he died from exhaustion. In the rectum and again above the sigmoid were irregular patches of membranous colitis. The membranes were greyish and leathery. (*e*) In a woman, aged 27, a suppurating ovarian cyst ruptured into the peritoneum; she died in five days: there were patches of membranous exudation in the caecum only. (*f*) A woman, aged 61, was admitted on February 2 for pyuria and cystitis. On February 6 diarrhoea and haematuria set in, and she died exhausted on the 14th. Just within the anus and extending for three inches up in the rectum was a large area of brownish-grey coagulation-necrosis, together with congestion and submucous haemorrhages as high as the splenic flexure. The whole thickness of the bladder was necrosed, and there were abscesses

in the kidneys. 1891.—(*g*) A man, aged 39, first taken ill on March 26, was admitted on April 2 for cellulitis of the neck; this was incised and much foul pus escaped. Death, April 9. Necrosis of thyroid cartilage, due to acute suppurative perichondritis, was found. In the ascending colon the mucous membrane was reddened, and there were several whitish exudation patches with a red border.

It is curious that this batch of cases of pyaemic membranous colitis occurred in the post-mortem room at Guy's Hospital during the years 1891, 1892, and 1893. Looking back for many previous years I do not find any other cases. We have already seen that in 1886 there was a batch of cases of septic non-membranous colitis. Dr. Goodhart has pointed out how in some periods pyaemia is frequently associated with infective endocarditis; and it appears also that at times it is liable to be associated with colitis either simple or membranous. It is not common in either case to meet with diarrhoea or other symptoms which point to implication of the colon; nor is it usual to find a general pyaemia, for in none of the membranous and in one only of the simple cases was there pus in the joints. In three of the seven membranous cases the kidneys were affected—two had abscesses, and one was in a state of non-suppurative inflammation; an interesting point, as one of the simple cases had a peculiar condition of kidney (*vide* p. 814). Five of the seven membranous and four of the five simple cases were in women; but perhaps this is to be explained by the liability of women to infection through the pelvic organs. The membrane usually has a distinct line of demarcation, and there may be several membranous areas separated by healthy mucous membrane. Some of these cases appear to indicate that a considerable amount of membrane may form in seven or eight days.

iii. Wilks and Moxon mention that membranous colitis may be met with in Bright's disease; so the simple, membranous, or ulcerative forms may complicate this malady.

iv. Bristowe says that membranous colitis may occur with pneumonia. The association of pneumonia with simple colitis was mentioned on p. 815.

v. A man, aged about 27, died under my care from diabetic coma. The mucous membrane of the first foot of the colon was acutely inflamed and had on it many patches of coagulation-necrosis. The inflamed area was sharply limited at one end by the ileo-caecal valve, but the limitation at the other end was indistinct. There was also membranous inflammation of the lower end of the oesophagus, and the stomach was much reddened.

Many authors describe diphtheritic colitis, but, as we cannot accept any membrane as genuinely diphtheritic until the Klebs-Löffler bacillus has been demonstrated in it, the existence of diphtheritic colitis is at present an open question. It is stated that membranous colitis may be found in persons dying from various other specific fevers, or tuberculosis. Fat necrosis of the peritoneum has been seen in cases of secondary membranous colitis. It is uncertain whether the *Bacillus coli* has anything to do with the production of this disease.

III. **Ulcerative Colitis.**—In the last edition the opinion was expressed in this article that although the colon is frequently ulcerated as a result of enteric fever, dysentery, tuberculosis, or malignant disease, yet cases occur in which this part of the bowel is extensively ulcerated quite apart from any of these diseases, and further, that at the bedside nearly all patients in whom this independent ulceration is found after death have, during life, presented such a grouping of symptoms as to enable us to predict that the colon will be found ulcerated. To this disease the name simple ulcerative colitis, or more shortly ulcerative colitis, has been applied. It is a bad name, because the colon is ulcerated in other diseases; but it is so generally used that much confusion would be caused by an alteration of it at present.

Since this article was originally published in 1897 it has been urged by Dr. Rolleston (56) and many others that this disease is really dysentery. First, it is said that the bacteriology of the two diseases is similar, but most of the work that has been done in the attempt to prove this has been carried out on cases of so-called asylum dysentery (Gemmel, Mott, Eyre, Vedder and Duval), and it may be that this epidemic disease is different from the sporadic cases of ulcerative colitis which we meet with in hospitals; and readers of the article on dysentery in this *System* (Vol. II. Part II. p. 477) will see that at present it is difficult to establish the claim of any disease to be dysentery on bacteriological grounds only. No allusion is made to any micro-organism in the definition of dysentery. Flexner (Vol. II. Part II. p. 490) says that the dysentery bacillus is not a hard-and-fast species, nor is it known whether the clinical varieties of dysentery depend upon the particular variety of bacillus. Further, he points out that dysenteric bacilli can be found in children who suffer from diarrhoeal diseases, and even in some healthy children. Other authors draw attention to the many varieties of dysenteric bacilli; Prof. Lorrain Smith tells us that dysentery is a disease due to more than one type of bacillus, and Prof. M'Weeney, criticising a paper by Prof. Saundby in which it was stated —according to M'Weeney quite wrongly—that the bacillus of dysentery had been found in a sporadic case of ulcerative colitis, points out that caution should be exercised in pronouncing a given strain of bacillus isolated from the contents of the bowel to be Shiga's or any other form that has received a name, so that even if the sporadic cases of ulcerative colitis had been fully investigated bacteriologically, it would at present be unwise for bacteriological reasons alone to conclude that ulcerative colitis and dysentery are the same disease. Turning to the clinical side of the question, some good observers (Osler (58), Allchin) are convinced that the ulcerative colitis here described and dysentery are different diseases, and therefore, as I think that this is probably true, I have decided to let the description stand; and since coming to this decision I am supported by Matthews, who from his experience of India concludes that "as far as clinical symptoms, treatment, and prognosis are concerned," the cases described by Prof. Saundby are not dysentery as known in the tropics. Future research will very likely shew that there are many

distinct diseases included under the names dysentery and ulcerative colitis, a supposition borne out by very different treatments advised for the several varieties of dysentery. We do not know the cause of ulcerative colitis; the diseases associated with it will be discussed presently.

Age and Sex.—The average age of eleven cases, of which I published an account (82), was 40 years. The youngest was 17, the eldest 59. In 1893 my house physician, Dr. F. J. Colman, collected 17 additional cases, and found the youngest was 21, and the eldest 54. Ulcerative colitis, therefore, is probably not a disease of childhood, nor of old age; and this conclusion is supported by Dr. Tooth's paper. It is not appreciably commoner in one sex than in the other; for out of 28 cases 13 were women and 15 were men.

Morbid Anatomy.—Any part of the large intestine may be affected, and generally the ulceration has no special distribution; but in one case it is said to have been most marked along the mesenteric attachment of the bowel: in another it appeared to follow the longitudinal muscular bands. One case, in addition to ulceration of the colon, shewed ulcers in the vermiform appendix. In an extreme case the muscular coat is exposed, and the ulceration is so extensive that only islets of mucous membrane are left here and there: often these are considerably swollen, and consequently they look taller than they otherwise would do; and frequently they are more or less stalked because of the ulceration which undermines them. The result of this is that a careless observer takes the islets of mucous membrane for polypoid growths, and the exposed muscular coat for the natural level of the colon. The colon may be much dilated, and where it is not ulcerated the muscular wall is sometimes hypertrophied. The peritoneal surface over the ulcer is usually normal; but it may be much congested, and if the floor of the ulcer is sloughing there may be a little local peritonitis around it. The vessels of the mucous membrane are dilated, and sometimes the membrane is black, as though from long-standing congestion. The number of ulcers varies from one or two to several dozens; but while ulcers of recent date are often more or less circular, and vary in size from a pea to a five-shilling piece, by the time death occurs their shape is usually very irregular; and occasionally, like all chronic intestinal ulcers, they shew a tendency to become transverse. When numerous they run one into the other, and produce a series of ulcerated surfaces so irregular in shape, that the comparison made by Bertrand and Fontan to the irregular pieces of a child's geographical puzzle is very apt. Generally at the time of the autopsy no sloughs are to be seen; but in a few cases one or two ulcers have shewn a ragged surface, indicating that a slough has been recently detached, and if the patient has died early in the disease they may be seen adherent to the ulcers. It is extremely rare to see any attempt at repair; in one case the account of the post-mortem examination states that in both the large and small intestine there were several pigmented spots of old ulcers, but there is never any evidence of constriction of the bowel. Sometimes perfora-

tion results: this may happen in any part of the caecum, colon, or rectum. In one case the floors of the ulcers were so thin that they bulged, forming little pouches. Rarely, as in cases recorded by Dr. Ormerod and Dr. Lazarus-Barlow, the perforations are very numerous. These numerous perforations, which are also seen in some cases of enteric fever, are difficult to explain; for as the patients, if we may judge by their symptoms, do not long survive the first perforation, there seems hardly time for the others to appear. Perhaps the explanation is that the peritonitis set up by the first perforation so softens the floor of the other ulcers that they rapidly give way. All the ulcers may be in an advanced condition, but if the patient die early in the course of the disease the mucous membrane of the colon is found to shew a highly vascular, soft, red surface covered with many recent ulcers. I have seen the whole colon in this state; there were more than a hundred superficial ulcers varying in size from a split pea to a threepenny-piece. When the disease is in this early stage the wall of the bowel is so softened that it is sometimes described as rotten. It may be possible in the same case to see all gradations between the early and late stages of the process; and even when the ulcers are of a considerable size and depth they may be still surrounded by a very red halo of congestion. There is never any satisfactory naked-eye or histological evidence that the process began by ulceration of the solitary follicles, although in a few specimens the follicles may be seen to be ulcerated, and in one case many of them contained a bead of pus; so unimportant, however, is follicular ulceration in ulcerative colitis that it was seen in none of the eleven cases I published in 1888. The difference between the two conditions will be given more fully when we come to speak of follicular ulceration. The mesenteric vessels are never mentioned as thrombosed; and the account of the post-mortem examination rarely states that the mesenteric glands are enlarged. It is clear from this description that it is often quite impossible to tell from looking at the bowel whether the patient died of dysentery or ulcerative colitis; but I have already pointed out the clinical reasons for separating the two diseases. In some cases the small intestine is affected, but this is rare, and the process is much more severe and apparently of longer standing in the colon. In one case there was a small ulcer in the stomach. The liver is often fatty.

Histologically the process is one of acute inflammation; first the mucous membrane becomes red, soft, and swollen, and may contain small haemorrhages. In the next stage there is a considerable accumulation of small cells in the submucous layer. This severe inflammation leads to necrosis of portions of the mucous membrane, which is got rid of either as small particles or as sloughs. In this way ulcers of various depths are produced, leaving the swollen islets of mucous and submucous membrane. Commonly the muscular coat is not much affected, but some increase of leucocytes may be seen in it. Frequently the only change in Lieberkühn's crypts is that the epithelium is cloudy and swollen; where there is ulceration they are cut off flush with the ulcer, unless it be so deep that they are

destroyed. Occasionally the upper part of the crypt gets blocked, with the result that the lower part is much distended.

Associated Diseases.—Most of the patients who die of ulcerative colitis shew no disease in any organs of the body except the intestines, and, if perforation has taken place, the peritoneum. The coincident diseases that in any way suggest a special association are Bright's disease, gout, and suppuration of the liver. The association of chronic interstitial nephritis with ulceration of the colon has long been known. Case 23 of Bright's "Tabular View of the Morbid Appearances occurring in 100 Cases in connection with Albuminous Urine" is that of a woman aged 40, in whom the kidneys were "hard, rough, and lobulated," and the large intestine was "ulcerated throughout." Mr. H. B. Robinson has recorded a case which shews the relationship very well, and another has been described by Dr. Bannatyne. Among the twenty-three cases of which I have notes of the condition of the kidney, chronic interstitial nephritis was present in six, and in at least four of these the renal change was advanced. One of the six patients was also suffering from lead poisoning, and two of the seventeen whose kidneys were healthy had urate of soda in their joints.

The association of abscesses in the liver with ulcerative colitis is rare. It is well known that small multiple pyaemic abscesses, accompanied by rigors and the usual symptoms of pyaemia, may occur when there is any source of infection in the periphery of the portal vein. But when we consider how common such sources are, it is remarkable that portal pyaemia is so rare. In thirty consecutive cases of ulcerative colitis I only found pyaemic abscesses of the liver once. Instances of large abscesses of the liver in association with ulcerative colitis have been recorded by Fagge and Dickinson (19), and the recent records of post-mortem examinations at Guy's Hospital contain another instance in a woman aged 30. Several years ago I had under my care a case of ulcerative colitis, in which the sequence of events was difficult to interpret; but it was very probable that the patient had both the large solitary form and the multiple pyaemic variety of abscess in his liver (84). Lastly, it may be that those cases of solitary abscess of the liver occasionally found in persons who have never been abroad, and who have never received any injury, are due to the fact that the micro-organism of ulcerative colitis has, like the amoeba of dysentery, reached the liver without causing any intestinal ulceration.

Symptoms.—It is nearly always the state of the bowels which seriously directs a patient's attention to his illness, and the first symptom which he remembers is usually abdominal pain, generally griping, sometimes very sharp and severe, and often sudden in its onset; in one case the patient shrieked under it. Soon it disappears only to reappear later; the duration of these painful attacks varies from a few minutes to many hours, and their alternate appearance and disappearance are very characteristic. The pain is always referred to the front of the abdomen; but its exact position varies in different cases, and also in different attacks

in the same patient. Occasionally there is also pain in the back and loins; and once I have known a patient complain of such severe pain in the front and sides of the chest that she was incorrectly thought to have pleurisy as well as ulcerative colitis. In acute cases the first attack may be excruciatingly severe; but often it does not cause much suffering; the intensity of the pain increases, however, in each succeeding attack. In the interval between the attacks the patient is usually quite free, or he may complain of a dull pain in the abdomen. It should be mentioned that the pain bears no relation to the ingestion of food, but is commonly worse when the bowels are open. The patient lies quiet on his back during an attack; but if perforation has occurred the legs may be drawn up. The mere presence of ulcers is insufficient to explain the pain, for it is commonly absent in enteric fever; it is probably due to some peculiarly irritating ingredient of the contents of the intestines which stimulates the nerves exposed on the floor of the ulcer, and thus sets up irregular peristaltic contractions. In the majority of cases there is no abdominal tenderness; when this is present it is rarely intense, and most often it is especially marked over some part of the colon.

The presence of severe diarrhoea—sometimes interrupted by short periods of constipation—is almost of equal symptomatic importance to the pain; it is often the first symptom noticed, and these two symptoms are never absent throughout the whole of a case. The frequency with which the bowels are open commonly varies between 2 and 11 or 12 times in the twenty-four hours; but in one of Dr. Sharkey's patients the bowels were open from 10 to 24 times in the twenty-four hours. A man aged 22, who died of ulcerative colitis in Guy's Hospital, had his bowels open the following number of times on each of the 14 days before he died, 6, 5, 2, 6, 7, 4, 5, 5, 8, 6, 6, 6, 5, 11. Although the act of defecation is often accompanied by abdominal pain, there is rarely the intense tenesmus characteristic of dysentery: nor have I heard of a patient complaining of that almost constant desire to go to stool which is so frequent in a severe dysenteric attack. If the case is mild an occasional solid motion may be passed; but the evacuations are nearly always fluid, dark, foul-smelling, and of a consistency varying between that of slime and water. So far as our present knowledge goes, they never resemble ordinary dysenteric or choleraic stools. Blood is very commonly present, especially after a period of constipation, sometimes as a mere trace, sometimes in large quantities; in the latter case the presence of much clot—when a patient describes the motion as being like red-currant jelly—forms an exception to the statement that the motions are fluid; but usually the blood is liquid, and is then bright red, shewing that the bleeding has been recent. Commonly it is passed with the motion, although not intimately mixed with it; and if the latter contain any solid particles small clots of blood will adhere to them. Occasionally it distinctly follows the motion, and rarely it precedes it. These differences probably depend upon the anatomical relation of the bleeding ulcer to the motion which is evacuated. The clots may be flat and smooth on

one surface and shreddy on the other, suggesting that they have been recently detached from the floor of the ulcer. In a few instances the patient was unaware that he was ill until he detected blood in his motions. Although after constipation a motion consisting of little but solid faeces may be passed, yet when diarrhoea is present very little faecal matter is seen in the evacuations, and what there is consists of a few small lumps scattered about in the fluid. In some cases a little mucus has been observed, but it is never present in large quantities, nor in the form of little transparent pellets, as in cases of follicular ulceration. Often shreddy masses looking very like sloughs are seen: they vary much in colour and size; under the microscope they are generally structureless, although occasionally leucocytes may be noticed, and there may be indications of epithelial cells; probably their structure has been destroyed by the action of the contents of the large intestine. In one or two cases a little pus is said to have been present in the evacuations, but this is certainly exceptional; enough pus to be easily recognisable is never seen. In one case, in Guy's Hospital, the *Amoeba coli* was found.

Soon the other symptoms are superadded to the pain and diarrhoea. The most frequent of these, and one of the earliest, is vomiting. Often when patients are admitted into hospital they say they have vomited incessantly, or have brought up everything they have taken; but usually under rest in bed and careful dietary the vomiting becomes much less. Sometimes it is unimportant; some patients indeed never suffer from it. I have known the vomit to contain blood in a case in which the ulceration was confined to the large intestine; the blood came, no doubt, from the rupture of some small vessel in the stomach, throat, or gullet. Neither the vomiting, the constipation, nor the diarrhoea stand in any proportion to the area of ulceration, but, like the pain, are to be regarded as due to the irritation of nerves exposed on the floor of the ulcer. The loss of fluid by vomiting and diarrhoea is probably the cause of the thirst, which may be a great trouble. Nausea may be complained of, either without vomiting or in association with it. The tongue is usually covered with a dirty white fur; but as the disease progresses it becomes red and dry with a brown fur. If he be seriously ill the patient has the drawn face characteristic of abdominal disease, and occasionally there is considerable sweating. The abdomen is commonly distended, and the peristaltic movements of the intestine may be visible. A rectal examination should never be omitted, because it is sometimes possible to feel the ulceration; in one case, which occurred before ulcerative colitis was a recognised malady, the surgeon, feeling an ulcer, concluded that the patient had malignant disease of the rectum or sigmoid flexure, and performed left lumbar colotomy. At the post-mortem examination on a patient under my care in 1893, one large and several small ulcers, of sufficient age for one of them to have extended through the internal sphincter into the perirectal connective tissue, were found just inside the anus. During life the diagnosis of ulcerative colitis was obvious, but I had examined the patient's rectum three weeks

before death, when the whole of the mucous membrane which could be reached by the finger was found intact. The ulcerative process may, therefore, extend rapidly.

The general symptoms are such as would be expected. The patient gradually becomes wasted, anaemic, and excessively weak, so that he lies in bed looking bloodless, sallow, and extremely ill. The pulse is feeble, and haemic murmurs may be heard. Irregular pyrexia is often present, the temperature ranging between 100° and 102° F.; but towards the end of the illness, if the patient be very collapsed and exhausted, it may be subnormal. The highest temperature I have seen was in a girl who was in the Hospital from October 24 till her death on November 12; her temperature was always between 102° and 103°, often over 103°, and twice it reached 104°. A girl in the Hospital in 1890, under Dr. Goodhart, had a temperature which ranged between 102° and 104°; and Dr. Sharkey gives a case in which 104·6° was attained. Towards the end of the patient's life there may be muttering delirium and other symptoms of the typhoid state. The cause of death is usually exhaustion, and in some cases it is accelerated by haemorrhage. Occasionally perforation of an ulcer is responsible for the result; and in one patient no perforation could be found although acute peritonitis was present; as the intestinal inflammation was very severe, it appeared probable that the peritonitis was due to direct extension of the inflammatory process through the walls of the gut. Although, as will be shewn directly, ulcerative colitis may be associated with granular kidneys, sufferers from it do not often shew signs of uraemia.

Prognosis.—Although patients thought to be suffering from ulcerative colitis are occasionally discharged cured, still the prognosis is very grave—so grave, indeed, that it is quite likely that most of these patients were not suffering from ulcerative colitis at all; this diagnosis has sometimes been confidently made when post-mortem examination has shewn the bowel to be only reddened. On the other hand, even fatal cases occasionally shew an attempt at repair of the ulcerated surface. Therefore, all we can say is, that while certainly the mortality is very high, it is not yet proved that the disease cannot end in recovery. The shortest case I know was in a man who died collapsed soon after admission to the Hospital. He said that three days previously he had been suddenly taken with severe diarrhoea and abdominal pain. In the colon were several large acute ulcers. At first sight it might be thought that the acuteness of this case was due to the presence of eight or ten similar ulcers in the small intestine from the duodenum downwards; but another case, in which ulcers were found high up in the small intestine, does not support this view. Occasionally patients state that they have been liable to diarrhoea for a long time—in one case as long as two years—but this is quite exceptional; the patient is usually dead within eight weeks from the commencement of the illness. Symptoms which especially indicate a speedy end are great tympanites, much loss of blood, profuse diarrhoea, and a high temperature.

Diagnosis.—Dysentery may occur at any age, but ulcerative colitis is not seen in children or the elderly. I have already said that the violent tenesmus, the burning pain at the anus, the constant desire to go to stool, and the scanty meat-washing evacuations which are characteristic of dysentery help to separate it from ulcerative colitis. Much more mucus is passed as a rule with dysentery than with ulcerative colitis, but vomiting is probably much less common in dysentery, which, however, is more painful than ulcerative colitis. Then, too, there is the lack of a history of residence abroad and of dysentery contracted there. If it be ultimately established that dysentery is due to a particular variety of micro-organism, the presence of this in the motions may help in diagnosis; but it is quite possible that as the morbid anatomy of ulcerative colitis is much the same as that of dysentery, the former disease may be due to another variety of micro-organism. It is highly probable that a point of practical distinction between the two diseases may be that ulcerative colitis is hardly ever as acute as an acute case of dysentery; while on the other hand it is never so chronic as are most of the cases of chronic dysentery that we see in England. It is stated that often attempts at repair of the affected bowel may be seen in dysentery, but this is very rare in ulcerative colitis; and swollen lymphatic glands, although often seen in those dead of dysentery, are hardly ever noticed in those who have succumbed to ulcerative colitis. On the other hand, as Matthews points out, perforation, which is not infrequent in ulcerative colitis, is very rare in bacillary dysentery. Indeed, consideration of what has been written since the first edition of this article was published leads me to believe that what is here described as ulcerative colitis is, as was maintained in the first edition, distinct from bacillary dysentery.

In actual practice the difficulty of diagnosis is usually to separate ulcerative colitis from malignant disease of the large intestine, from intestinal obstruction, and from some form of primary anaemia. A mistake is usually due rather to faulty examination of the patient than to any real difficulty. Fortunately the form of intestinal obstruction most likely to be mistaken for ulcerative colitis is intussusception, which, however, is very rare at the age at which ulcerative colitis is most common (*vide* also p. 797). I have known the severity of the anaemia in a case of ulcerative colitis suggest some primary blood condition. I have never heard of enteric fever simulating ulcerative colitis; but one patient, who also had severe bronchitis, was at first thought to be suffering from tuberculosis; a correct diagnosis, however, was soon formed. Once I saw ulcerative colitis overlooked in a patient who had a large abscess in his liver; the diarrhoea from which he suffered was supposed to be due to the leakage of the abscess into the intestine. However, he was moribund when seen. It is worth while to repeat that as the ulceration often extends low down in the rectum, a rectal examination should never be omitted; if necessary a speculum, or a sigmoidoscope, should be used; an anaesthetic may be necessary; either should be passed with care, for fear of rupturing an ulcer. It may be well also to warn

the physician against a diagnosis of syphilis in such a case; in syphilis the anus is always implicated.

Treatment.—Unfortunately this often avails but little. The patient must remain in bed and be kept warm. If the pain be very severe, hot abdominal fomentations are comforting. The diet should consist of milk, custard, jelly, blancmange, and similar articles of food: vegetables and fruits should be avoided, and beef-tea will probably increase the diarrhoea. Three or four ounces of brandy every twenty-four hours are generally useful. Bearing in mind the abdominal pain, and that the diarrhoea is often excessive, opium is probably the best drug to prescribe. In a severe case the patient should be kept well under its influence; it matters little what preparation is given. As a rule a grain every four hours may be ordered to begin with, and if this does not suffice the dose may be gradually increased; sometimes a starch and opium enema is more efficacious in checking the diarrhoea than opium by the mouth. Of course, if the patient be suffering from interstitial nephritis it will be necessary to use great caution in prescribing opium; in any case it is doubtful whether it is wise to push it to such an extent as to cause actual constipation. Half or three-quarters of a pint of a saturated solution of boracic acid at about 100° F. may twice a day be allowed to run slowly into the bowel through a long rectal tube introduced as far as possible, while the hips are well raised. Considering, however, the extremely intractable nature of the disease, it may be justifiable, in some cases, to make an artificial anus just above the caecum, to let the faeces be discharged through it, and then to syringe the whole of the large intestine from the artificial anus to the natural anus with boracic solution twice a day. I saw, with Mr. Monier-Williams who has recorded the case, a patient for whom this was done, and it apparently saved his life, and G. C. Macdonald has recorded two cases. The artificial anus can be closed later. Some make an opening in the appendix, and irrigate the large bowel through that, employing either plain water, boracic solution, or a solution of nitrate of silver (20 grains to the pint). Short-circuiting is rarely justifiable, for the ulceration extends to the rectum in a severe case. Operative treatment has been especially recommended by Pauchet and Prieur.

To relieve the vomiting a drop of tincture of iodine in half an ounce of water, or three drops of dilute hydrocyanic acid in an ounce of an effervescing mixture, may be given every hour. If these fail, trial may be made of ten grains of each of bicarbonate of sodium and carbonate of bismuth suspended in an ounce of water with sufficient mucilage. If the rectal haemorrhage is dangerous, an ounce of liquor ferri perchloridi diluted with three ounces of water should be injected per rectum as far up the colon as possible. If this fail, possibly a weak solution of adrenalin would succeed.

Asylum Dysentery.—Diarrhoea accompanied by the passage of much blood and mucus is common in asylums. There were 1155 cases in the London County Asylums in the four years 1901 to 1904: of these, 266

were fatal. The symptoms and post-mortem appearances of asylum dysentery need not be described, as they are so similar to those of bacillary dysentery. Many authors, on the assumption that asylum dysentery was the same disease as the sporadic cases of ulcerative colitis, have concluded that this is the same as bacillary dysentery. Reasons, however, have been given which prevent our being very sure that ulcerative colitis is the same as bacillary dysentery; further, although asylum dysentery is so common in asylums, yet, as Dr. Knobel points out, it is so rare in other institutions, *e.g.* prisons and infirmaries, that great doubt must attach to the assumed identity of asylum dysentery with the sporadic cases of ulcerative colitis met with in hospitals—indeed these are so few that if the two were an identical disease we should have to admit that it was almost confined in this country to asylums, where it is the cause of from 4 to 5 per cent of all deaths, although bloody and mucous diarrhœa is very rarely fatal among the adults of the community at large. A few years ago Dr. Mott and others considered that its spread in asylums was due to direct infection. Dr. Knobel doubts this, for he says that in spite of increased precautions in the way of isolation and disinfection there has been practically no diminution of the disease. Many have tried to associate asylum dysentery with a definite dysenteric bacillus, but it is difficult to see how any bacillus, *e.g.* Shiga's, can be the sole cause, seeing that in some countries it causes widespread epidemics, and asylum dysentery is limited to asylums. Indeed, it is difficult to resist the conclusion that an essential cause of the disease must be in the asylums, and probably Dr. Knobel is right in believing that the nervous disease present in the insane lowers the trophic resisting power of the intestine, so that organisms which in health could not produce ulceration now can. He also points out that the frequent appearance of the disease in new asylums suggests that turning the soil has some influence. We have seen that patients with ulcerative colitis frequently have chronic Bright's disease; over 50 per cent of those who die from asylum dysentery have it, and many have atheroma. The best treatment is probably rest in bed, milk diet, and irrigation of the bowel with warm water with or without salol, permanganate of potassium, or boracic acid dissolved in it.

IV. **Follicular Ulceration.**—This condition is far more common in the large intestine than in the small. It begins with an accumulation of small round cells in the solitary follicles, which therefore swell. Soon the epithelial covering dies and ruptures, and then there follows disintegration of the follicles, the result being many small round ulcers, beginning as a minute depression in the centre of the follicle, and having sharply-cut edges, perhaps a little overhanging, but never bevelled. These ulcers do not extend deeply, although they may extend laterally a little beyond the follicle; for the process of disintegration usually advances a little way in the submucous coat round the follicle, and the ulcer then becomes flask-shaped. In a well-marked example the gut is quite honeycombed by these circular ulcers, which vary in size from a

hemp-seed to a large pea. I have not met with any case in which there was cicatrisation: in fact, none of the cases in our autopsical reports is mentioned as healing; nor is perforation ever recorded, the floor of the ulcer being always the muscular coat. Occasionally ulcers are described as running together to form an irregular ulcerated surface, but this is not common. The mucous membrane between them is not usually affected, except that in their immediate vicinity there is a considerable collection of small cells. Sometimes at the post-mortem examination a bead of pus will be found in some solitary follicles, and if the patient had lived this minute abscess would have burst, and a follicular ulcer would have resulted; but it is by no means certain that all follicular ulcers are preceded by suppuration.

The patients in whom follicular ulceration is found have always died of some other disease, often of the gastro-intestinal tract; thus I find that of ten adults one had dysentery, one cancer of the rectum, one membranous colitis, one enteric fever, and one a femoral hernia. Of the other five, three were the subjects of tuberculosis, but none of them happened to have tuberculous ulceration of the intestine: one died of multiple sarcoma, and the fifth of a retroperitoneal malignant growth. Of the ten adults seven were males, and the ages varied from 17 to 54. Follicular ulceration of the colon occurs about once in every 500 necropsies at Guy's Hospital. It is never diagnosed during life, for the symptoms of the fatal disease quite overshadow any that might be attributed to the ulceration. Occasionally it is seen in the small intestine, and there it does not differ from the ulceration as it occurs in the colon. Follicular ulcers are rarely found in cases of ulcerative colitis; when they are present they should be regarded as a complication, for there is no reason to believe that ulcerative colitis begins particularly in the follicles.

Follicular ulceration is more frequent in children than in adults. Thus, Holt met with follicular ulcers in the intestine in twenty out of seventy fatal cases of infantile non-tuberculous diarrhoea. They were never seen in cases that had lasted less than a week, and the proportion of follicular ulceration was highest in those cases which had lasted more than ten weeks. He found that cases with considerable follicular ulceration ran a slower course than other varieties of diarrhoea, and that marked pyrexia and blood in the stools were exceptional. In two of the twenty cases the ulcers were in the small intestine only; in fifteen, in the colon only; and in three, in the colon and small intestine. When in the small intestine they were always near the caecum. The commonest seat for the ulcers was the descending colon and sigmoid, and in this region they were most numerous, largest, and deepest. In children as in adults it is very rare to find any evidence of healing in follicular ulcers. Softening in the centre of a follicle often occurs as an early post-mortem change in the intestines of children: a depression forms, and the condition may be mistaken for early follicular ulceration.

It is common in persons dying of various diseases, especially in the colon, to find the follicles enlarged, swollen, and prominent without any

ulceration. I have noticed that they are especially well marked in fatal cases of exophthalmic goitre.

In children the solitary follicles of the intestine in the normal state are hardly visible to the naked eye; but in the large intestine they are often seen projecting above the surface: and they may have a black dot in their centre, presenting the well-known shaven beard appearance. This is especially common of children who have died of diarrhoea.

V. **Vascular Ulceration.**—This may be *venous or arterial.*

With regard to the *venous*, I cannot do better than quote from Wilks and Moxon, who say: "Ulceration of the large intestine is one of the most common post-mortem appearances we meet with; in persons long ill with various visceral complaints we often find large ulcers in various parts of the large bowel, but more especially in the caecum, ascending colon, and sigmoid flexure. These are mostly of a chronic character with raised indurated edges; some spreading, while others are healing. They probably have the same pathology as many ulcers on the leg connected with a retardation of the venous circulation. These ulcers have their length in the direction of the transverse course of the bowel, passing around its circumference. In cases of phthisis and tuberculous disease it is not uncommon to find such ulcers, and not apparently having a tuberculous character."

As the portal circulation is impaired in cirrhosis of the liver, it is not surprising to find venous ulcers of the colon in this disease. In 1896 a man died of cirrhosis of the liver; an ulcer was found in the colon 3 feet 6 inches above the sigmoid flexure. I find that at Guy's Hospital these venous ulcers, shewing no trace of growth or tuberculosis, are also found chiefly in persons who have died of phthisis or malignant disease. Thrombosis of the mesenteric veins affects the small intestine as well as the large, causing much reddish lividity and swelling of the mucous membrane, which may go on to ulceration.

There are at least three varieties of ulceration of the colon produced through the *arterial* system.

The mesenteric vessels may be blocked by emboli, and thus ulceration may complicate infective endocarditis. In a well-marked case the embolism leads to gangrene of the bowel, haemorrhage into its swollen coats, and dilatation of the veins; and the ulceration which follows may lead to perforation. Although peritonitis usually supervenes even if no perforation has taken place, I was once much struck with the fact that while the interior of the bowel was gangrenous there was no local peritonitis. The main symptoms are intense abdominal pain and tenderness, and, if the patient live long enough, there may be tympanites and foul stools containing blood: but it is remarkable that severe results of obstruction of the mesenteric vessels may be found post-mortem, although no symptoms have been present during life.

In the second variety ulceration of the colon may be caused by thrombosis of the mesenteric artery or of one of its branches. The following are good instances of this; the first was a remarkable case

under Sir Henry Howse:—A woman, aged 49, had gangrene of one of her legs. The post-mortem examination shewed thrombosis of the corresponding femoral artery, the aorta, and the superior mesenteric artery; peritonitis, a communication between the small intestine and ascending colon, and another between the small intestine and descending colon. The descending colon contained numerous small pigmented ulcers; in the caecum there was an extensive ulcer, and in the small intestine many sloughy ulcers were found. A woman, aged 66, with mitral stenosis, had complete thrombosis of the inferior mesenteric artery. The colon and last five feet of the ileum were intensely engorged and ecchymosed; this change would probably have gone on to ulceration. The pressure of tumours on the mesenteric arteries may lead to ulceration of the intestine.

The third variety of arterial ulceration is that which depends upon imperfect circulation through the mesenteric arteries, owing to atheroma of the aorta. The following case is a good instance of this:—

In a man, aged 66, admitted for gangrene of the legs, most of the vessels were extremely atheromatous, and the atheroma of the aorta much diminished the orifices of the mesenteric vessels. The colon, which had a worm-eaten appearance, was extensively ulcerated.

VI. **Haemorrhagic Ulceration.**—In some diseases submucous extravasations of blood take place, and these may break down and lead to ulceration. Haemorrhagic ulcers are usually small, shallow, rounded, and discrete, although occasionally two or three may run together; submucous haemorrhages may commonly be seen in their neighbourhood.

Persons suffering from chronic Bright's disease are liable to haemorrhages in any part of the body; and it is known to those who make many post-mortem examinations that these patients occasionally present ulcers in the intestines, or even in the stomach. Wilks and Moxon drew attention to the fact thirty years ago. It may be, as Dr. Dickinson suggests (20), that submucous haemorrhage is one cause for the ulceration. He gives eight cases in which submucous haemorrhages in the intestine were found in chronic Bright's disease, and the liability of these patients to haemorrhages was demonstrated by their presence in the retinae of all those whose eyes were examined. Submucous haemorrhages may be associated with ulcers in the colon, as is proved by Dr. Dickinson's and other cases. For instance, in 1888 a man died under the care of Dr. Taylor; the kidneys were granular, there were numerous superficial ulcers and submucous haemorrhages near the caecum and in the ascending colon. But after hearing Dr. Dickinson's paper, I could not help feeling that, although some intestinal ulcers found in the subjects of Bright's disease are very probably haemorrhagic, yet all could not be thus explained; for it must be remembered that, as Wilks and Moxon have taught for many years, enteritis and colitis are not uncommon complications of this disorder, and we might fairly expect that sometimes before death they would proceed to ulceration. Then, again, ulcerative colitis is often associated with Bright's disease, and in a severe case the swelling

of the mucous membrane and the whole intensity of the process, which may lead to the denudation of several feet of mucous membrane, shews that the ulceration cannot have originated in submucous haemorrhages. They may be present near the ulcers, it is true, but surely they ought sometimes to be regarded more as evidence of the intensity of the inflammation than as the cause of the ulceration.

Submucous haemorrhages, leading to ulceration, may occasionally occur in other general diseases—such, for instance, as purpura, scurvy, and severe forms of anaemia. In a case of Addison's disease there were some small intestinal ulcers, and the intestinal mucous membrane shewed pigmentation.

VII. **Trophic Ulceration.**—Dr. Acland, Mr. Targett, and I have raised the question whether disease of the central nervous system can cause ulceration of the colon, and our cases are given in full in the first edition of this *System*. (See also Asylum Dysentery, pp. 834, 835.)

There is a specimen in the Guy's Hospital Museum shewing enteritis of the lower part of ileum. The inflamed part is thickened and contracted, the mucous membrane is covered with granular lymph. All the coats of the bowel are infiltrated with small round cells. The patient was a woman aged 35, who was admitted under Dr. Pitt for haematemesis and severe anaemia. At the autopsy the left lobe of the cerebellum was softened, there was a large chronic ulcer in the stomach, and a few patches of superficial ulceration in the colon. There is also another specimen of numerous small round ulcers which occupied the whole of the colon. In some the muscular coat is exposed; in others a thin pellicle covers the floor. This specimen was taken from a man, aged 51, who was admitted for fractured spine in the lumbar region with complete paraplegia. He died fourteen days after the accident, having had diarrhoea for some days before death. In another case of fractured spine in the lower dorsal and lumbar region, the patient lived for two months, and after death a mild degree of colitis was found and one superficial ulcer in the colon. Blood was passed during life. A patient who during life shewed symptoms of locomotor ataxy and ulcerative colitis, died in Guy's Hospital. The spinal cord shewed the changes characteristic of locomotor ataxy, and the intestine those of ulcerative colitis. I have seen another patient with undoubted tabes and all the symptoms of ulcerative colitis.

These cases should direct our attention to the state of the colon or the small intestines in diseases of the nervous system. Seeing the ease with which bedsores and cystitis occur in lesions of the spinal cord, it is hardly surprising that ulcers are occasionally found in the bowel.

Dr. Cowan has called attention to the frequent occurrence of enteritis, colitis, and intestinal ulceration in the insane, and is inclined to regard the intestinal changes as, in some cases, secondary to the nervous disease; and Dr. Knobel considers that asylum dysentery is partly due to deterioration of nerve-cells affecting the trophic nerve supply to the colon. But according to Dr. Eurich the postulate is by no means proved: he lays

great stress on the general lowered vitality of lunatics, rendering them an easy prey to disease of all sorts. Most of his cases in which the condition of the colon was not due to some recognised disease, either had gangrene of the lungs, and so might have had septicaemia, or they were filth-eaters.

VIII. **Dilatation of the Colon.**—Cases in which the large intestine is dilated may be divided into four groups: (i.) The first contains those in which the distension is entirely gaseous, is not due to any obstruction, and is only one symptom of some other illness. It is often seen in association with peritonitis and enteric fever, and it may occur with almost any severe illness. Usually there is at the same time some distension of the small intestine. When extreme, it is not only distressing to the patient, but it is of great importance, for it adds considerably to the danger of the original disease; it greatly hampers the movements of the heart and lungs, thus causes palpitation and difficulty of breathing, and, if it occur in association with heart disease, is particularly dangerous. In an extreme case the abdomen is very distended and tense; it hardly moves on respiration; it is hyper-resonant on percussion; the tympanitic note extends well into the flanks; the liver is pushed up so that the hepatic dulness does not reach to the lower margin of the ribs, and the splenic dulness is obliterated. If the distension or tympanites, as it is usually called, affect one part of the intestine much more than another, the shape of the distended part may be visible. The patients are nearly always constipated; they do not usually pass much flatus by the rectum, nor by the stomach; but they complain of borborygmi, which may be audible to bystanders.

The gas in the intestine, according to Bertin, consists of carbonic acid and hydrogen, and Hoffmann, quoting from Planer and Ruge, says that marsh-gas is also frequently present. At the post-mortem examination all that is observed is that the intestine is distended, and that its walls are consequently stretched and thin; sometimes the distension is enormous, for the colon has been stated to be as big as a man's thigh. The cause of the dilatation is probably paralysis of the muscular coat of the bowel. The only difficulty of *diagnosis* likely to occur is in the distinction of this condition from those rare cases in which there is gas in the peritoneal cavity.

The *treatment* of tympanites is difficult and uncertain; and its appearance is very unwelcome. Various carminatives, especially the aromatic oils, are usually recommended. They are conveniently given in drops upon sugar; but although I have often given them I have never seen them of any use in serious cases of distension of the large intestine. Enemas give a better chance of success; turpentine and asafetida enemas sometimes afford relief, frequently they fail. In other cases a pint of soap and water, or half a pint of warm olive oil, may be injected up the rectum, but as with carminatives, so with enemas, we properly shrink from much use of them in enteric fever or peritonitis. Any of these means may be supplemented by hot fomentations applied to the abdomen.

The attempt is often made to draw off the gas from the bowel by passing a long tube up to the sigmoid flexure, but this usually fails.

Puncture of the bowel has been recommended, and this operation is commonly done in veterinary practice. There is a description of it as performed upon sheep in Hardy's *Far from the Madding Crowd*. Bertin mentions a case in which it was performed fifty times on the same patient without the least accident. Still, it is a proceeding which should rarely be attempted. A very fine trocar and cannula, which have been previously boiled, should be used; and after the trocar has been withdrawn, and while the gas is escaping from the cannula, the abdominal walls should be gently pressed so as to make them follow the intestine as it collapses. The puncture is best made in the middle line, for here the abdominal walls are very thin; and into the transverse colon, as this is usually much distended. The benefit is rarely permanent, for the gas quickly gathers again. Acupuncture should never be employed, as it may lead to the escape of gas into the peritoneal cavity.

(ii.) The second group contains those cases in which the distension of the colon is due to some solid substance within it. In the human subject it is excessively rare for the distending substance to come from outside the body; but concretions consisting of a little faecal matter incorporated with vegetable fibres, hair, and oak-husk have been found, and a good deal of phosphate of lime is usually precipitated from the contents of the intestine, and this sometimes makes the concretion as hard as a stone. These stony masses are found in the intestines of many vegetable-feeding animals, especially perhaps of horses, cows, and goats. They constitute one variety of bezoars, a name applied to all calcareous concretions found in animals. They are much commoner in the small intestine than in the large. The substances not introduced from without may be gall-stones or impacted faeces. The former may be as large as a hen's egg, and usually get into the duodenum—or more rarely directly into the colon—by means of a fistulous communication with the gall-bladder (*vide* p. 743).

Distension of the colon by faecal matter is of great clinical importance, as it is of common occurrence. The faecal mass, which is often of very large size, is usually in the sigmoid flexure or in the descending colon. I have known a mass in this position mistaken for a large tumour of the kidney. The subject of distension of the colon from foreign bodies is fully discussed in the articles "Constipation" and "Intestinal Obstruction."

(iii.) The third group contains those cases in which the dilatation of the colon is due to some organic obstruction in front of the dilated gut. In these the intestine hypertrophies as well as dilates, and the hypertrophy is almost entirely of the circular fibres of the muscular coat; but the longitudinal may be somewhat more uniformly spread and more numerous than usual. This muscular hypertrophy may render peristalsis easily visible; but this appearance, though common in the small, is very rare in the large intestine. The intestinal contents retained behind the stricture usually set up some colitis, which may proceed to ulceration, when the

well-known "distension ulcers" are produced. This is a bad name, for there is no reason to suppose that the mere distension causes the ulcers. The colitis is shewn by swelling and injection of the mucous membrane, and in severe cases the inflammatory process advances so far that the mucous membrane is dark and sloughy. The ulcers may consist of nothing but a slight loss of substance of the epithelial surface; or the whole intestine for several feet behind the obstruction may be covered with irregular ulcers, as was the case with a women in whom, as a result of malignant disease of the rectum, "the whole of the colon above the stricture was distended and worm-eaten by small ulcers." These, as they increase in size, come to have ragged edges and run one into the other, producing a large area of ulceration. Often the muscular coat is exposed, and sometimes even perforation of the serous coat with consequent fatal peritonitis takes place. The rapidity of this inflammatory ulcerative process depends much upon the completeness of the obstruction and the nature of the retained contents. Sometimes it is very rapid, as, for instance, in the case of a woman who was admitted for intestinal obstruction of nine days' duration. At the autopsy volvulus of the sigmoid and acute colitis were found; the distended sigmoid was covered with irregular ulceration on its mucous surface; two of the ulcers were deep and nearly perforating. This case, however, is exceptional, for distension ulcers are commonest in association with chronic obstruction, and are, therefore, most frequently met with behind malignant growths or impacted faeces. The ulcers themselves when exposed appear to have existed for some time, and they may even be pigmented. It is curious that they may be a long way behind the obstruction without any ulcers in the intervening bowel; thus, in one case they were found only in the caecum and ascending colon, although the obstruction was due to carcinoma of the sigmoid. This colitis may, after the obstruction has been overcome, be difficult to treat, and is the cause of the severe diarrhoea which sometimes follows when after long-standing faecal impaction the bowels have at last been opened. It will subside, however, under rest in bed, careful diet, warmth to the abdomen, and, if necessary, an occasional starch and opium enema.

The dilatation behind an obstruction may lead to considerable distension of the abdomen. By careful watching of the peristaltic movements, by percussion, and by palpation, we can often make out whether it is the large or the small intestine which is dilated; but it must not be forgotten that a dilated small intestine may be as large or larger than the colon, and often lies transversely in the abdomen. On the other hand, a distended transverse colon may bend down towards the pubes like a coil of small intestine, and the sigmoid may be so distended as to reach quite over to the right side of the abdomen. Considerable or even extreme dilatation of the colon is not uncommon in the insane. I need hardly add that slight distension often occurs in persons of a constipated habit.

(iv.) The fourth group contains those very rare cases of so-called idiopathic dilatation of the colon, often known in Germany as Hirsch-

sprung's disease; Dr. Hawkins considers neuropathic dilatation and hypertrophy the best name. We have had only one case at Guy's among the last 16,000 post-mortem examinations. It is difficult to be precise as to the number of cases recorded, for it is doubtful whether some were not long-standing cases of faecal impaction. This criticism applies particularly to Bristowe's first case, for the patient, a girl aged 8 years, had always been subject to constipation, and her bowels had not been open for seven weeks before admission. On her death the large intestine was enormously dilated from the caecum to a point within 2 inches of the anus, and was completely filled with faeces. Perhaps also Peacock's case of a man aged 28, always constipated, who had had a bad attack of faecal impaction at 17, and in whom hard faecal accumulation had been broken up more than once, belongs to this group. In him also the large intestine was very distended, being 6 to 8 inches in diameter; it contained fifteen quarts of a semifluid greenish faeces, and the mucous membrane was much ulcerated. In this case the abdominal distension, "which made him appear like a woman just before her confinement," was associated with displacement of the heart and liver upwards, with oedema of the legs, penis, and scrotum, and with albuminuria—these last symptoms being apparently due to pressure on the vena cava or renal veins.

When writing the first edition of this article I got together 12 cases; Crozer Griffith, writing in 1899, had found 23 cases in literature and recorded one himself. Kredel, writing in 1904, thinks the condition more frequent than is commonly supposed, and alludes to many cases, but I much doubt whether some are instances in point. All authors are agreed that the condition is far commoner in males than in females, about five out of every six cases being males. Putting aside the cases which may have been due to faecal impaction or other obstruction, a well-defined group stands out among those which remain, namely, those in which the sigmoid flexure only is distended. The following are examples of this:—Dr. Herringham and Mr. Bruce Clarke give the case of a man over 70 who had always been extremely constipated. Six years ago he had had an attack similar to the present, but it had been overcome by strong purgatives. For the last six years he had had little or no trouble in this way, but his bowels had now not been open for eight days. The abdomen was distended, enemas and purgatives failed to relieve him, and he died of perforation. The sigmoid flexure was found enormously distended, and resembled a dilated stomach. Its walls were hypertrophied, and its inner surface ulcerated and gangrenous. There was more than one perforation; the rest of the intestine was normal. Constipation lasting eight days can hardly be regarded as sufficient to have produced this condition of the sigmoid. Sir John Banks's patient, a man aged 50, was found to have the sigmoid flexure so distended that, bent on itself, it filled the abdomen; it was 2 feet in circumference. In Chapman's case, a male deaf-mute aged 58, the sigmoid bent on itself filled the abdomen; its circumference was 14 inches. It had actually got up between the right lobe of the liver and the diaphragm. Money and

Paget's case shewed the sigmoid to consist of two large sacs even bigger than an ordinary dilated stomach. Dr. Gee's first case was that of a boy aged 4½, in whom the sigmoid was found to consist of two huge sacs lying vertically side by side, one in the right half of the abdomen, the other in the left. The greatest circumference of the sigmoid was 13 inches. His second case, also in a boy, was very similar. The distinguishing characteristic of these cases is that the sigmoid is enormously distended, filling the whole of the front of the abdomen. It is in shape like a dilated stomach, and, in Chapman's case, it is stated that the longer curvature measured 20 inches, the smaller 10 or 12. The view adopted by some speakers, when Dr. Herringham and Mr. Bruce Clarke's paper was read, was that a slight accumulation of faeces at one part of the sigmoid leads to kinking of it; that this increases both the obstruction and the accumulation of faeces, and thus one reacts on the other. But this explanation is not certain, and several questions will have to be answered before it is accepted. Why, if there is thus an obstruction at the lower part of the sigmoid, is this part of the bowel so enormously distended and, in these six cases, the rest of it so little distended or not at all? Then, again, these patients were not excessively constipated. For instance, although Dr. Herringham and Mr. Bruce Clarke's patient had been of a constipated habit, it so happened that before the fatal distension of his sigmoid, "for the last six months he had had little or no trouble, the bowels had been open regularly without more than an occasional mild aperient." When seen he had only been suffering from want of action of the bowel for eight days. Then we must remember that though obstruction about the sigmoid and rectum from faecal impaction or growth is very common, the condition we are describing is very rare; and although constipation is much commoner in women than men, yet this dilatation of the sigmoid is almost confined to males, some of whom are so young that the trouble can hardly be secondary to constipation; and from many of the descriptions it appears that the distended sigmoid was not so full of faeces as we should have expected if there had been obstruction, and in many cases there was diarrhoea. Sir F. Treves has recorded a case in which great dilatation was due to congenital narrowing of the rectum, and believes that all cases of "idiopathic" dilatation of the large bowel can be explained by some form of obstruction other than faecal impaction; but all other writers are opposed to this view, for usually no obstruction is found after death, and as many of the patients are young, a commonly accepted view is that there is a congenitally abnormal development of the colon, in virtue of which it is longer and perhaps wider than it should be, therefore it hangs over into the pelvis and forms a loop, especially if the meso-sigmoid is long; the faeces and gas collect in this loop and distend it, often some colitis is set up and even diarrhoea occurs—the colitis may lead to ulceration and even perforation. But, as Dr. Hawkins points out, this explanation is unsatisfactory, for much-folded pelvic colons are common while dilatation is rare, and may be associated with a colon which is short. Others have

thought that this local dilatation of the sigmoid is comparable rather with the cases of dilatation of the stomach without obstruction of the pylorus. And I notice that Dr. Rolleston and Mr. Haward adopt the same view. The one is often associated with dyspepsia, the other with faulty action of the bowels; but in both the precise relationship of the perverted action to the dilatation is obscure, and in neither is it easy to be sure of the cause of the condition. Why the sigmoid should be more affected than any other part of the bowel is not clear to us unless there is a developmental defect; but it must be remembered that, according to some authors, such as Trastour, it is the habitual store-house for faeces between the acts of defecation, and that slight dilatation of it is common.

The cases in which the dilatation was near the sigmoid rather than of it, probably belong to the same clinical group; the following are examples:—One, recorded by Mr. Walker and Mr. Griffiths, was that of a boy aged 11, whose abdomen was enlarged and tympanitic soon after birth; his bowels were regular at first, but afterwards constipated, although they could always be opened by enemas, and it does not appear that there was ever any obstruction. His appetite was bad, his breathing was short, wind would occasionally "roar away," and then the distension lessened. So distended was he, that although his height was only 4 ft. the abdomen measured 23 inches from the ensiform cartilage to the pubes, and its greatest girth was 3 ft. 11 inches. On opening the abdomen it appeared filled with an enormous coil of intestine, looking like a flexed thigh and leg, formed by the transverse and descending colon, the latter 23 inches in circumference. The sigmoid and ascending colon were somewhat enlarged. The diaphragm was so pushed up that from the top of the diaphragm to the suprasternal notch was only 2½ inches. The peritoneal coat of the affected bowel was thick, the muscular coat was hypertrophied, and there was no submucous tissue; the mucous membrane was thick, often denuded of its epithelium, and in a state of chronic inflammation. A second case, also a boy, is that recorded by Dr. Rolleston and Mr. Warrington Haward. He was twelve years old at death, and he had suffered from attacks of constipation and vomiting together with a distended abdomen since the age of two months. The descending colon and the caecum were enormously dilated. Another case, recorded by Little and Callaway, was an imbecile male aged 34, who had always had an inordinate appetite; and after eating an unpeeled orange and a pound of Banbury cakes was taken with abdominal pain and sickness. Aperients acted, but the next day extreme distension was noticed, the circumference at the umbilicus being 45 inches. The heart was very much displaced, and he sank on the seventh day. There was enormous dilatation of the transverse and descending colon, and of the sigmoid, the muscular and mucous coats of which were very thick. A fourth case is recorded by Dr. Goodhart (34). The large intestine was dilated from the caecum to the anus, but the dilatation was not local, as in the other cases, nor was it so extensive. There was a considerable

amount of ulceration. Wells gives a case of an elderly man in whom the transverse colon was distended in the form of a loop, but it is just possible that some adhesions of the small intestine were the cause of this.

Prof. Osler's case (57) forms a connecting link between the group of dilatation of or near the sigmoid and those of dilatation of other parts of the colon. The patient, a coloured boy aged 10, was thin, but enormously distended in the abdomen. He was not particularly constipated, and often had diarrhoea and vomiting. Intestinal peristalsis could be seen. For a time washing out the bowel with water, introduced with a long tube when the hips were elevated, led to some relief; but this was slight, and the pain and vomiting continued. The abdomen was opened; there was no stricture. The sigmoid was 18 inches in circumference, the caecum was half this size, and the bowel progressively increased in size from the caecum to the sigmoid, which was folded on itself, but not so as to cause any obstruction. An artificial anus was made at the most prominent part of the sigmoid. The mucous membrane seemed normal, but the muscle was hypertrophied. After the operation his appetite and general condition improved, he had no tympanites, he gained in weight, and passed the faeces through the artificial anus. Dr. Morley Fletcher and Mr. Betham Robinson have recorded the case of a boy in whom the dilatation included the rectum and colon as far as the hepatic flexure. The patient died comatose unexpectedly.

Looking at the ages of the recorded cases it seems probable that we shall have to distinguish two varieties of so-called idiopathic dilatation of the colon; namely, that occurring in elderly subjects, and that, apparently due to some congenital inertness of the bowel, met with in young children. I think these distinctions are sufficiently striking to be of practical value, and that the cases in which the colon as a whole is affected are only a different degree of those in which the sigmoid alone is implicated. Further, some writers, *e.g.* Kredel, think that only a few of the sufferers attain adult life, and they form the group of elderly patients, and it may be that if one of these patients can attain adult life he will manage to get on to old age, and thus the children and elderly adults really form one group. Dr. Hawkins agrees with this, and that the inertness is a congenital neuro-muscular defect in virtue of which the bowel cannot pass its contents on. This difficulty may be increased in infants by spasm of the anus. Mr. Brooke records a case aged 21 in which there was an undoubted congenital history. The chief symptoms of this condition are as follow:—

Symptoms.—The distension of the intestine is very great, and percussion shews that it is chiefly due to gas. R. H. Fitz has suggested that many of the cases of chronic phantom tumour recorded by the older writers were examples of this form of dilatation of the colon. Shortness of breath is a cause of complaint, and the patient may be livid, because the diaphragm is pushed up and breathing is hampered. The upward displacement of the heart gives rise to palpitation. The splenic dulness

is obliterated, the hepatic dulness is diminished, and the liver cannot be felt. The abdomen is always distended, sometimes enormously so, and then it may be tympanitic all over; distinct coils and peristaltic movements are sometimes, but not always, visible. Often the huge distended bent sigmoid may be seen. The distension is greatest in the left iliac fossa. Slow alterations in shape may be visible. Pain and vomiting are usually absent. In Money and Paget's case the intra-abdominal pressure was so great that the legs, scrotum, and penis were swollen, and the patient had albuminuria. After the gas had been let off by puncture of the intestine the albuminuria passed away, the specific gravity of the urine fell, and the patient passed eight pints of urine in the night. Dr. Gee's first case had haematuria shortly before death. Most of the patients for some time, often for years, have had some difficulty with the bowels, constipation supervening from time to time, the bowels not being open for many days, occasionally not for a week or two, and in one case at least not for a month; but constipation is not usually very serious, and yields to purgatives or more often only to enemas. In Money and Paget's case the bowels had always been regular; and for some years before the fatal attack they had been so in Dr. Herringham and Mr. Bruce Clarke's case. Occasionally diarrhoea sets in shortly before death or it may alternate with constipation. Flatus is often passed and faeces may be drained away through a tube. The condition found after death has already been indicated in the description of the individual cases. Both layers of the muscular coat of the gut are hypertrophied, and its mucous membrane is often ulcerated, probably from the colitis set up by the irritation of the retained decomposing faecal matter. Except that the kidneys may be hard, like heart kidneys—probably owing to compression of the renal veins—the whole body, save the colon, appears healthy; and in no case which has been used as a basis for this description has any obstruction been found in the intestine. The small intestines are collapsed and lie behind the colon. The condition appears, therefore, to be purely local. Very rarely tetany is associated with it, and Dr. Langmead has recorded a case in which several attacks of tetany were due to a huge dilatation of the sigmoid. The distended bowel contains semi-solid or fluid faeces and an enormous quantity of gas; solid faeces are exceptional; in a man aged 23 the colon and its contents weighed 47 lbs. (Formad). It has been found on measurement to be increased in length as well as in diameter. Sometimes there is much fever, many patients are very wasted; a boy ten years old only weighed 48½ lbs. The outlook for the younger patients is very grave. Crozer Griffith says that only three out of the twenty-four cases he collected reached adult life, but it may be that slight cases which do not see a doctor grow up and form the elderly group to which we have alluded. Death may be due to perforation of the ulcers, but often the cause is not clear; no doubt death is often due to the colitis. Often it took place a few days after the patient was first seen, and with some it was unexpectedly sudden.

Enemas and purgatives commonly had no effect; and even if the bowels were opened the patient was not much improved thereby. The passage of a long rectal tube often failed to bring away any gas, and although sometimes this proceeding or puncture of the intestine relieved the pressure for a time, the gas soon reaccumulated. As these results are so poor I suggested in the first edition that the best chance is afforded by opening the bowel above the dilated part; and as this part is usually the sigmoid there would be very little difficulty in the operation. For some time the faeces should all be passed through the artificial anus, and the distended sigmoid should be thoroughly flushed from the artificial opening to the anus every day with clean tepid water. Much improvement followed in Prof. Osler's case, in which the sigmoid was opened. In Crozer Griffith's case a right inguinal colotomy was done, but the patient died. In Sir F. Treves's, Mr. Clutton's, and R. H. Fitz's cases the distended bowel was successfully excised. Dr. H. P. Hawkins believes that most cases can be successfully treated if operated on early, and he much prefers anastomosis of the iliac part of the sigmoid flexure with the pelvic part when the sigmoid only is affected, but as after this operation the dilated portion of the sigmoid has subsequently undergone a volvulus, it should be fixed. In one of his cases operated on by Mr. Makins, the upper part of the pelvic colon, just below the anastomosis, underwent enormous dilatation. In Dr. Morley Fletcher and Mr. Betham Robinson's case the abdomen was opened and pounds of faeces were pressed by the hand through the anus. The patient improved much and remained well for nearly two years but then died. They suggest that this treatment should be tried before more severe surgery. In their case it appeared especially suitable, because the dilatation reached so near to the anus that no anastomosis could have got below it. In the discussion which followed the reading of this paper some considered simply draining the dilated colon the best treatment.

IX. **Sacculation of the colon** is not common. The diverticula are usually the size of a pea, but I have seen them big enough to receive the little finger, and half an inch in depth. They are most frequent in the descending colon, sigmoid flexure, or upper part of the rectum. Usually they are very numerous, are placed close together, and contain a little faecal matter. They are described as hernial protrusions of the mucous membrane through the muscular coat, and no doubt this suggestion is correct; but often a thin layer of muscle is spread over the whole surface of a sacculus except just at its bottom, which consists of mucous membrane and peritoneum only. Sometimes, as in a specimen I shewed at the Pathological Society (83), these diverticula lead into appendices epiploicae; and Wilks and Moxon mention a case in which they protruded between the layers of the mesentery. Perhaps they should be regarded as due to obstruction in the form of chronic constipation; and in favour of this is that they are found for the most part in elderly people, and, so far as I know, never in children. As a rule they give rise to no symptoms; but we have in the Museum at Guy's Hospital a specimen in

which perforation of the fundus of one of these sacculi led to the formation of an abscess between the left kidney, spleen, and ascending colon.

Solitary diverticula are very rare; but in 1888 an autopsy was made at Guy's Hospital on a patient who died of malignant disease of the oesophagus; and in the colon ten inches from the ileo-caecal valve was a diverticulum half an inch long which admitted a No. 10 catheter.

This is perhaps the best place to mention that the appendices epiploicae may become pedunculated, and enlarged at their lower part; so that they really form subserous lipomas. Small pedunculated lipomas may become detached, and may be found free in the peritoneal cavity. They are sometimes calcified. I have known a foreign body work its way from the sigmoid into one of the appendices and form an abscess, and Mr. Bland-Sutton has described similar cases.

X. **Laceration of the Colon.**—Sometimes, but happily very rarely, this is due to an enema tube or a bougie. There are two such specimens in the Guy's Hospital Museum. In the first case an enema was given by means of a long tube to a man aged 75; shortly afterwards he was admitted to the Hospital collapsed, and he died the next day. A laceration was found at the lower end of the sigmoid flexure; the perforation in the mucous membrane was only one-eighth of an inch in diameter. The other specimen, presented by Hodgkin and Callaway, represents a condition that could hardly occur nowadays. It consists of the sigmoid flexure with a rent more than an inch long, due to a bougie which had been passed, under the impression that the patient had stricture of the rectum; he was really suffering from faecal impaction. I have heard of the sigmoidoscope being passed through the wall of an extremely ulcerated bowel. A nurse or a doctor may be alarmed by the apprehension that the perforation of the rectum has been produced by an enema, as in elderly people the evacuation of the bowel by an enema is often accompanied by much faintness and even by syncope. The pulse may become quite small, and the patient appear collapsed. I was once summoned very urgently to see an old lady in this condition, who had been allowed to get up to go to stool after the enema. This should never be permitted in elderly or feeble people who are unaccustomed to enemas. In fact, constipation in such persons is to be specially avoided.

The kinds of accident that most frequently damage the intestine are, being run over, kicked in the abdomen, or stabbed there. The small intestine is much more often affected than the large; but some years ago a man who had been run over was admitted into the Hospital, and the only lesion found was a large tear in the ascending colon. The laceration may not go quite through, as in the case of a man who was run over by a brewer's dray. The small intestine was torn completely across, but in the descending colon the muscular and serous coats were extensively lacerated, although the mucous membrane was entire. We have a specimen also in which the muscular coat only is ruptured, and another in which the serous alone is affected.

XI. **Perforation of the Colon from without.**—Abscesses in a variety of positions may burst into the colon. Thus a bullet entered the abdomen from behind; a large retroperitoneal abscess formed and opened into the descending colon. Instances of iliac and psoas abscesses rupturing into some part or another of the colon might be quoted; and a suspicion of what has happened may be afforded by the disappearance of the tumour and the passage of pus by the bowel. I have come across two cases, both in women, in which the localised collections of pus, which occur in suppurative peritonitis when adhesions are numerous, had burst into the colon. In both there had been many perforations, and in both the opening inwards of the abscesses had caused a number of clean-cut circular ulcers, in the floor of which was a perforation. In the second case there were other ulcers as well, so it is probable that the presence of pus in the colon set up colitis followed by ulceration. I have seen a localised peritoneal abscess pointing in the inner surface of the stomach; in a day or two it would have formed an ulcer there with a perforation on its floor. Abscesses in connexion with the appendix may burst into the bowel, probably much more often than is thought. More than once I have known an appendicular abscess burst into the rectum.

Hepatic abscesses may burst into the bowel. Murchison gives a case in which a large abscess opened freely into the colon, and there was extensive ulceration of the adjacent portion of the ascending colon. This is important, as, like one case just mentioned, it goes to shew that pus discharging into the bowel can set up ulceration of it; but still it is possible that this case was one of ulcerative colitis, associated with a large hepatic abscess. In rare cases abscesses formed in connexion with the kidney may open into the colon; and Bright recorded the case of an abscess of the spleen discharging into the colon. Habershon mentions two remarkable cases of cystic ovarian tumours: in one the tumour opened into the ileum, and thus became filled with faeces; in the other it suppurated, and ruptured both externally and into the caecum, so that there was a discharge of faeces externally. Fagge mentions a case of suppuration about a growth in which the same thing happened; and Habershon gives an instance in which an abscess in the abdominal parietes burst both externally and into the colon, and so formed a fistulous communication into the large intestine.

Hydatids of the liver may be discharged into the colon, and then cysts may be found in the evacuations. The gall-bladder may adhere to the hepatic flexure, and large gall-stones may ulcerate their way into the bowel. Occasionally in cases of ulcer of the stomach, either simple or carcinomatous, this organ may become adherent to the transverse colon into which the ulcer perforates, and thus a gastro-colic fistula is formed. In 1887 this condition was found in a woman who died from chronic bronchitis. As far as was known it gave rise to no symptoms, but cases have been recorded in which such a fistula has caused faecal vomiting.

XII. **Malformations of the Colon.**—Malformations of the colon are rare, but by far the most common are those which depend upon mal-

positions of the caecum. It will be remembered that in intra-uterine life this is first outside the abdomen, then inside just under the umbilicus, which is then near the pubes. In its next position it is on the left of the abdominal cavity; its last position on this side is near the cardiac end of the stomach; then it passes across the abdomen and comes to lie under the liver, and finally it descends to the right iliac fossa (see also p. 587). The caecum in an adult may be in any of these successive positions, from just inside the umbilicus to the normal one in the right iliac fossa. It is quite possible that in some cases the persistence of the caecum in a part of the abdomen, which in the natural course of events it should have left before birth, is due to fixation from intra-uterine peritonitis—a disease by no means uncommon, especially in children afflicted with congenital syphilis. When the caecum is fixed in its abnormal position the large intestine continues to grow, and consequently various abnormal loops are formed. As these conditions have been known to give rise to difficulties both to the physician and to the operating surgeon, they should never be forgotten in considering a case which is manifestly obscure. Various references to actual cases are given by Mr. Lockwood, who points out that the failure of the caecum to descend is often associated with an undescended testis. Other malformations are excessively rare. Mr. Lockwood quotes two cases in which, for part of its length, the large intestine consisted of two tubes; and Guy's Hospital Museum contains a specimen taken from a boy aged seven days which shews a membranous septum completely occluding the bowel at a point nine inches above the anus. The colon was much dilated above; below, the gut was contracted to the size of a cedar pencil.

XIII. **Metallic Pigmentation of the Colon.**—Sometimes lead may be deposited in the tissues of the colon. The three following instances have occurred at Guy's during recent years:—A man who had lead colic died in 1888 from chronic Bright's disease. The whole of the caecum and large intestine was very black, and the line of demarcation at the caecum was quite sharp. Sir T. Stevenson found the colon to contain 0·0086 per cent of lead. Dr. Pitt recorded the case. Another man was admitted in 1890 for saturnine epilepsy; he died, and the colon was found to be very black from lead. Microscopical examination shewed minute black granules arranged just outside the vessel in the vascular loops. A woman who was under my care in 1892 for lead colic died from saturnine eclampsia. At the ileo-caecal valve there was a sharp line of demarcation beyond which the colon was almost black as far as the sigmoid. The pigmentation was most marked at the mouths of the follicles, and was in places patchy. There is nothing to shew that lead is not deposited in the colon by the same process as leads to the formation of a blue line on the gums.

Two cases of black colon from mercury occur in the *Pathological Society's Transactions*. In one recorded by Dr. C. T. Williams, a lady aged 74 took eight grains of calomel a week all her life. At the autopsy the internal surface of the large intestine was remarkably black in patches.

The discoloration began abruptly at the ileo-caecal valve. Analysis shewed the presence of mercury in the mucous and submucous coats. Another is recorded by Dr. Rolleston: a man aged 64 had undergone antisyphilitic treatment for some time, how long is not quite certain. The caecum and colon were uniformly pigmented. There was no pigmentation of the small intestine. Analysis shewed the mucous and submucous tissues to contain mercury. The colon may be found blackened from bismuth in patients who have taken much of it during life, and probably, like mercury and lead, it is deposited in the tissues of the large intestine; but analyses on this subject are wanting.

XIV. **Malignant Disease of the Colon.**—As a patient afflicted with malignant disease of his colon nearly always suffers from intestinal obstruction, malignant disease of the colon is for the most part described under that head (see p. 748). Occasionally, however, the growth is soft and medullary; then it breaks down in the centre and intestinal obstruction does not arise. A necropsy in these cases reveals that several inches of colon are affected with growth, usually forming an obvious tumour visible directly the abdomen is opened. On cutting into this mass it is found that the normal channel of the intestine is transformed into an elongated cavity with black, ragged, sloughy walls, but passing into healthy colon both above and below. I have known the whole of the transverse colon to be converted into such a mass. The contents of the cavity consist of a foul mixture of faeces, blood, and necrotic fragments of growth. If the sigmoid or caecum be affected this mass may grow into the subjacent bone; if it is in other parts of the colon it may extend into the stomach, liver, or kidneys. This state of things during life may give rise to no symptoms, as in the case of a woman who died under my care from cancer of the liver. During life the primary seat of the growth could not be found, but at the autopsy it turned out that the sigmoid flexure was a sloughing carcinomatous mass. On the other hand, it may be easy during life to detect a tumour, especially if it be in the transverse colon or the caecum. The patient may pass large quantities of foul, dark grey or black fluid motions, in which perhaps fragments of growth and small blood-clots are seen. Sometimes between the passage of such motions the bowels may be constipated. The patient suffers pain, he wastes, and I have known death to occur in such a case before any symptoms of secondary growths in the liver appeared. When the tumour is in the caecum or sigmoid it may cause oedema of one leg and thrombosis of the external iliac vein, and when it is in the transverse colon we must carefully distinguish it from an enlarged liver, carcinoma of the greater curvature of the stomach, or an omentum puckered up by some form of chronic peritonitis. The implication of other organs by direct spread of the growth rarely gives rise to any symptoms by which it can be recognised. I have known the glands above the left clavicle enlarged secondary to carcinoma of the large intestine.

It is obvious that in most instances the only treatment for these cases,

in which there are no signs of obstruction, is to deal with symptoms as they arise. It is very rarely that the case is seen early enough for the surgeon to be able to excise the growth. For fistula in connexion with these growths see p. 754.

Sarcoma of the colon is very rare; Jopson and White have collected 22 cases; it may be either round- or spindle-celled. *Lymphadenoma* of the intestine is mentioned elsewhere, p. 577; it is excessively rare in the large intestine, but we have a specimen in which it occurred as a part of generalised lymphadenoma. A mass the size of a man's fist was growing in the caecum, and numerous masses projected from under the mucous membrane in the colon.

Secondary growths are occasionally seen in the colon, usually on its peritoneal surface; they rarely give rise to symptoms. Malignant disease may spread from the stomach to the colon, as described in the article on "Tumours of the Stomach," p. 507. The inroads of cancer from other organs are of little interest in this place.

XV. **Tuberculosis of the Colon.**—Wilks and Moxon point out that tuberculous ulceration of the ileo-caecal valve is frequent, and that tuberculous ulceration may be found in the appendix. The trouble occurs in no other part of the intestine than the caecum in about 8 per cent of the cases of intestinal tuberculosis. The colon is not infrequently affected with tuberculous ulcers exactly the same as those seen in the small intestine. They are commonest near the caecum. [For chronic Hyperplastic Tuberculosis *vide* p. 760.]

Polypi are not uncommon in the colon, but they are of no clinical importance. These and other innocent tumours of the colon resemble those of the small intestine described on p. 575.

Specific diseases (as dysentery, enteric fever, etc.), in which the colon is affected, will be found under their appropriate headings. Lardaceous disease of the colon does not differ from that of the small intestine.

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THE DIFFERENTIAL DIAGNOSIS OF DISEASES OF THE ANUS AND RECTUM

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THESE diseases, strictly speaking, belong to the sphere of the surgeon. Nevertheless, the physician is often consulted in cases of alleged diarrhoea, in which an exploration of the rectum may be of the first importance;

or the presence of piles may hinder the cure of cases of anaemia or other ailments which properly fall to the care of the physician.

The maladies which affect the anus and rectum are of great variety and are widely prevalent; they cause the sufferer much pain and discomfort, and are the source both of local annoyance and of mental distress. Nevertheless, in spite of the importance of the subject, the diagnosis of anal and rectal diseases has not received the great attention which is due to it. Various reasons appear to deter some medical men from investigating these ailments with sufficient care, especially when the patient is a woman. Thus, no examination is made at all, or but a very perfunctory one. The consequences of this neglect would be serious enough if there were but one rectal malady; they are intensified by the number of the diseases affecting this small portion of the human frame.

A careful local examination is absolutely necessary; first of all to arrive at the cause of the suffering, and then to determine on the right line of treatment to be employed for the cure of the particular malady. It is one of the greatest mistakes in medical practice to imagine that Unguentum gallae cum opio and sulphate of magnesium are the panacea for all rectal troubles. Each disease requires its proper mode of treatment, otherwise there will be no cure; indeed the patient may be made still worse. But if the diagnosis is correctly made and appropriate treatment used, whether medicinal or operative, an ordinary case of rectal disease can be cured readily and with little fear of a recurrence. Unfortunately, such success will never be attained so long as all rectal troubles are ascribed to piles; indeed, in some instances medical treatment for piles has been pursued when the patient was actually suffering from malignant disease. For such shameful carelessness no censure can be too strong. Again, even if the ailment be piles which are continually bleeding, patients are sometimes told that the haemorrhage will do them good; though they may be gradually growing weaker from more or less loss of blood. It would be just as reasonable to tell a patient that bleeding from the urethra or lungs was beneficial. This habit of regarding piles as the one malady of the rectum is found also in our patients when they come for consultation. Patients tell us that they are suffering from this complaint; their own diagnosis is too often accepted, and a thorough examination is postponed, with consequences more or less serious if the case really be one of fissure, polypus, fistula, ulceration, or even of cancer. We can only arrive at a correct diagnosis by making a thorough examination. First of all, however, the patient may be asked some questions in the following manner; further interrogation may follow a careful local examination:—

The patient should be asked if he has any pain, and if so, when and where; an affirmative answer may refer to fissure, ulceration, abscess, or malignant disease respectively.

The patient should be questioned as to the presence of a discharge. The discharge of pus will confine the diagnosis to abscess, fistula, and ulceration. Most patients are, however, unable to distinguish between

pus and mucus. Mucus will suggest piles, ulceration, or malignant disease. Blood may be discharged in any rectal ailment, and is therefore of no aid in diagnosis. Nevertheless it is an important symptom calling for careful investigation.

Inquiry should next be made as to the presence of any protrusion at stool, and whether it has to be returned by pressure. A protrusion may arise from piles, polypus, or polypoid growths.

The presence of diarrhoea or constipation should next be investigated. If diarrhoea, is it in the early morning on first rising? Is any blood or slime passed with the stools? Diarrhoea will indicate ulceration or cancer, morning diarrhoea cancer in particular.

From the replies to the above questions we may easily obtain some idea of the nature of the disease. But a diagnosis of rectal disease based upon symptoms only is most untrustworthy. The symptoms of rectal disease vary so greatly in different cases that a diagnosis made from the symptoms without a local examination is quite valueless.

The next step is a thorough examination. There are various positions for this; and if the case be a difficult one to diagnose, the patient may be required to kneel or even to stand up. But the best and at the same time the most delicate posture is for the patient to lie on the side, with the knees fully drawn up to the abdomen and the buttocks well to the edge of the couch, and for the examiner to kneel on the floor facing the buttocks. The patient should be asked to hold up the upper buttock with the left hand so as to give a good view of the anus and the surrounding parts. The medical adviser should then look carefully round the parts. His first attention should be turned to the condition of the skin of the anus, to see whether it is cracked, inflamed, or eczematous; or whether there are any scratch-marks. He should note the shape of the anus, and observe whether it is pouting, appearing to be tightly contracted, and so forth. Moreover, he should not fail to look for any orifices of fistula, and should not disregard the presence of any excessive amount of loose skin around the verge of the anus.

This ocular examination completed, with the forefinger of the right hand he should diligently feel round the anus for at least two inches from its orifice for the purpose of discovering any tender spot. Drawing the finger over the parts, he should press deeply in order to detect any induration pointing to the site of an abscess, or any cord-like ridge running up towards the anus, indicating the track of a fistula. The pressure may cause pus to ooze from the sinus of a fistula, or from the anus itself. Any small folds of skin must be carefully separated; for between or behind them the orifice of a fistula may be situated, and this may be easily overlooked. When any one of these abnormal conditions round the anus has been observed or felt, the next step is to separate the verge of the anus upwards and downwards with the fingers of both hands. At the same time the patient should be directed to strain down, as this action will give a view of the interior of the anus for about half an inch. In this manner a fissure may be noted and can be readily examined; the situa-

tion of a fissure in the large majority of cases being in the middle line behind: or again piles or a polypoid growth may protrude.

Then follows the most important point of a thorough examination, the insertion of the finger into the bowel. The finger, well lubricated with some unguent, is gently inserted, the patient straining down as before. This act releases the sphincters and renders the insertion more easy. At first merely the tip of the finger is placed within the entrance, and the breadth and tightness of the external sphincter are carefully ascertained. Then the finger is inserted a little farther and a diligent scrutiny is made of the interval between the two sphincters; it is in this space that piles are to be felt as enlarged rectal folds; and here, too, in at least 90 per cent of cases, the internal openings of fistulas are found as small dimples or as areas of induration. These internal orifices of fistulas are as a rule not more than an inch from the anal margin; it is altogether a mistake to suppose that their situation is high up the rectum. Again, between the sphincters we may find polypoid growths, the frequent cause of fissure. Unless these are discovered and removed it is useless to treat a fissure, as they will prevent it from healing.

When this area has been explored the finger is passed a little higher up the rectum, and the internal sphincter is searched for any induration which might point to the existence of an internal fistula, or of an abscess. Last of all the finger is pushed to the full extent into the bowel and the higher parts are carefully examined. Several points are to be noted, namely, the prostate gland towards the front, or the uterus which is easily to be felt. Or again, we may discover a malignant mass projecting into the upper part of the rectum, and this will give the finger the same sensation as is caused by the os uteri when felt from the vagina. If the symptoms appear to indicate a polypus the finger must be gently swept round the bowel; by this plan the stalk may be felt and the polypus itself may be brought within reach. When the finger is withdrawn, note should be taken of the character of any discharge left upon it, such as faecal matter, blood, pus, and so forth. Great care must be taken to avoid causing pain, as a proper examination cannot be made if the patient is moving about. The finger should be inserted slowly, and all movements should be as gentle as possible. The so-called "ballooning of the rectum" is of no value in diagnosis. It is due to distension of the rectum with air or gas, and is a purely accidental circumstance. If a patient be examined in such a position that the abdominal wall is at a lower level than the anus, the passage of the finger into the rectum will in most cases cause ballooning by allowing air to enter. Apart from this, it often occurs in patients who are flatulent or are the subjects of excessive alimentary fermentation.

When this thorough examination has been made with the finger, starting from the outside and passing upwards as high as possible in the order described above, and when all the different points with regard to normality or abnormality of state have been carefully observed, something further may still be needed to confirm or to complete the diagnosis. If

about or near the verge of the anus there is a suspicious orifice, suggestive of a fistula, a probe may be inserted with the greatest gentleness into this orifice, made to find its way quietly along the hardened ridge towards the anus, and thus passed into the bowel; unless the sinus be too tortuous. Again, with the patient straining down, and the physician everting the anal margin with the fingers, a probe may be used gently to explore any ulcer or fissure, in order to discover whether there be any burrowing backwards and outwards, or even running up the bowel from the fissure. This burrowing often exists, and if it is not detected the patient will not be cured either by the use of ointment, or by the division of what is presumed to be a fissure, but which is in reality a fissure with a small sinus running from it. The probe may be employed in a similar manner if the finger light upon an orifice situated between the two sphincters. Should there be an induration around the anus, which would prove this orifice to be the opening of a fistula, there would be no necessity to use the probe; but if there be no such induration, the probe must be introduced along the finger of the left hand as far as the orifice, and then passed into it to discover any sinus which starting from it may run up the rectum.

After a careful digital examination, it is in many cases necessary to examine the rectum with a speculum. A great deal of practice is, however, necessary to be able to use a speculum properly and without causing pain. The majority of specula are worse than useless, either because nothing can be seen through them, or because from their construction they cause pain when being inserted or withdrawn. A small fenestrated speculum with thick edges for examining the anal canal and a modified form of Kelly's short proctoscope for examining the upper part of the anal canal and lower rectum are, however, useful instruments if properly used. A good electric forehead-lamp or hand-lamp is a necessity with either. With the former, fissures or ulcers can be examined, and with the latter, piles, polypi, or growths in the lower rectum can be seen.

Internal piles cannot always be felt by a finger in the rectum, as being soft they convey no distinct sensation to the finger. They may usually be seen by getting the patient to strain down while the buttocks are separated, or a suitable speculum will reveal their presence.

It is possible that all this examination may reveal nothing; but the symptoms may appear to indicate some disease higher up the bowel, namely, the presence of a growth, one of the forms of ulceration, or a fibrous stricture. If nothing has been found in the rectum to account for the symptoms, or if the local condition seem insufficient, a careful and thorough examination of the higher part of the bowel must be carried out.

Examination with the Sigmoidoscope.—The bowel must be well emptied with aperients and enemas, and then examined with the electric sigmoidoscope. This instrument does not cause any pain, and an anaesthetic is not necessary except in special cases. With it the whole of the interior of the rectum can be examined, and, further, it can be passed into the



Fig 1



Fig 2



Fig 3



Fig 4



Fig 5



Fig 6

PLATE I

FIG. 1.—Cancer in the sigmoid flexure. 14 cm. from the anus. Removal attempted per abdomen, but operation had to be abandoned on account of secondary glandular infection found on opening abdomen.

FIG. 2.—Carcinoma in the middle of the sigmoid flexure. Man, age 49.

FIG. 3.—Ulcerative colitis, shewing superficial shallow ulcers on the mucosa of the sigmoid flexure, and adherent flakes of white mucus; mucous membrane granular. Man, age 32.

FIG. 4.—Ulcers in the mucous membrane at the junction of the rectum and sigmoid flexure. Woman, aged 40, with chronic constipation.

FIG. 5.—Cancer in the sigmoid flexure seen through the sigmoidoscope. 22 cm. from the anus. Resected per abdomen, and end to end anastomosis performed. Man, age 59.

FIG. 6.—Cancer on the anterior wall of the sigmoid flexure at the brim of the pelvis. An elderly man. The tumour could not be detected by any other method.

sigmoid flexure and the greater part of this segment of the bowel explored. In this way acute and chronic inflammation of the bowel can be examined, ulcerative conditions can be seen, and their limits and severity gauged. It also enables us to detect tumours, malignant or otherwise, which are out of the reach of the examining finger. If a tumour be detected in the higher portion of the bowel its mobility may be estimated and its position in the bowel ascertained with considerable accuracy. If necessary, small portions of the growth can be removed through the instrument for microscopical examination. A further advantage is that this instrument makes it possible to diagnose tumours in an early stage before they have become fixed and while their successful removal by operation is still feasible. It is also of great value in the diagnosis of ulcerative and catarrhal conditions of the bowel. In all cases of diarrhoea associated with the passage of mucus or blood in which a digital examination does not reveal sufficient cause for the symptoms, the sigmoidoscope should be employed. This instrument has entirely replaced the use of bougies for diagnostic purposes. It provides an accurate diagnosis in many cases in which formerly this could only be guessed at from a study of the symptoms; in short, it marks a great advance in rectal surgery. Like most special instruments it requires a certain amount of practice before it can be used successfully.

Syphilitic ulceration or stricture can only be diagnosed from the patient's history or from the presence of other syphilitic lesions. In cases of suspected tuberculous ulceration a careful bacteriological examination of the discharges should be made. If the case be one of traumatic stricture the patient has probably suffered from a very prolonged and severe confinement. In the dysenteric and bilharzial affections it will be found that the patient has resided abroad.

Lastly, attention should be turned to the abdomen; this should be examined with care, especially the left iliac fossa. The hand should be pressed deeply into it, and note should be taken of any tenderness, or of any tumour which may be felt. Sometimes, even when all these methods of scrutiny have been pursued, a sure diagnosis may still be wanting. When this is the case the patient must be anaesthetised and the examination must be carried out again in the various ways described above. Under the anaesthetic the abdomen should be thoroughly examined, and a bimanual examination of the rectum should be made with the left hand on the abdominal wall and the first finger of the right hand in the rectum.

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VISCEROPTOSIS

By Arthur Keith, M.D.

Synonyms.—*Enteroptosis, Splanchnoptosis, Glénard's Disease*

Introduction.—In 1885 Frantz Glénard (17) described a condition of sinking or prolapse of the abdominal viscera which occurred, to a greater or less degree, in nearly half (46 per cent) the patients undergoing a cure at Vichy. This condition he called *enteroptosis.* In *Les Ptoses Viscérales,* a monograph of nearly 1000 closely printed pages, he relates the circumstances leading up to his recognition of enteroptosis: (1) He was impressed by the frequency with which the transverse colon could be felt through the abdominal wall of the dyspeptic and neurasthenic as a narrow, contracted, cord-like body—a condition which he named *entérostenosis*; (2) on tracing the colon into the right loin, he was surprised to find that the right kidney was frequently displaced and palpable; (3) he also found the right lobe of the liver prolapsed into the right loin; (4) and that the stomach reached to or below the umbilicus. He conceived that in all such cases the viscera of the abdomen had undergone a pathological descent or prolapse, and that the displacement was the direct cause of many obscure symptoms. Glénard, however, has always maintained that, with the exception of a few traumatic or primary cases, enteroptosis is not a cause, but a result of a morbid condition; that prolapse of the viscera is not a disease, but a manifestation of malnutrition due to functional disorder of the liver.

Although the study of enteroptosis commenced in France, the majority of the publications on this subject have appeared in Germany. In 1881, four years before Glénard's original paper, Landau published his work on displacement of the kidney, and in 1885, his monograph on "floating liver" and the "paunch-belly" of women, but in neither publication did he recognise that he was dealing with part only of a general condition. Ewald proposed to replace the name "enteroptosis" by "splanchnoptosis"—a change which has much in its favour; for in complete and true enteroptosis there is not only a displacement of the abdominal, but also of the thoracic viscera. During recent years contributions to our knowledge of enteroptosis have been made in Germany by Lindner, Israel, Meinert, Hufschmidt, Küttner, Ott, Kumpf, Hertz, Huber, Schwerdt, Langerhaus, Stiller, Obrastzow, Frickhinger, Becker and Lennhoff, Rosengart, Aufrecht, Dennig, Koellreutter, Klatt, and Weissmann; the last-named gives a most useful survey of the German literature. In France Féréol, Récamier, Faure, Chapotôt, Hayem, Terrier and Auvray have written on this subject; and in America rapid progress has been made in the diagnosis and treatment of enteroptosis, as may be seen

in the publications by Einhorn, H. A. McCallum, Rose and Kemp, Beyea, Clarke, and Dolley, and others (58). In England the subject has not received the attention it deserves. In the present article I propose to incorporate the main conclusions of the writers cited above, and to give the results of my own inquiries, some of which have been already published (27, 28).

The Normal Position of Abdominal and Thoracic Organs.—In estimating the degree of displacement of abdominal organs in any given patient it is essential to have accurate and easily applied surface-markings of the normal position of these viscera. The most important of these surface-markings is a line drawn across the body at the junction of the sternum with its ensiform process—a point easily detected by the depression felt beneath the sternal insertion of the seventh pair of costal cartilages. This line—the *sterno-ensiform line* or plane—marks the normal upward level or height of the abdominal viscera, which here fill the right and left domes of the diaphragm. On the right side, and near the mid-clavicular or nipple line, the dulness or opacity of the liver should reach the sterno-ensiform plane in the supine position, and fall some 10 mm. (half an inch) short of it when the patient stands up. On the left side of the body, the resonance or translucency of the stomach does not reach the sterno-ensiform line; in the prone position it falls 10 mm. below it; in the upright posture, 15 mm. short of it. In the mid-line of the body the central tendon of the diaphragm, separating the liver from the heart, lies 10 mm. below the sterno-ensiform line. The sterno-ensiform line is the base or standard from which all measurements indicating the degree of ptosis are taken. There are certain degrees of variation in the upper level of the abdominal viscera; in healthy and apparently normal individuals the domes of the diaphragm may be found 25 mm. (1 inch) below the standard given here; but if the depression be more than 25 mm. ptosis of the viscera is certainly present; in all cases in which the depression was over 35 mm. enteroptosis was present in its most severe form.

The sterno-ensiform line, marked on the patient by a narrow strip of lead-foil for radioscopic examination, or by a pencil line for examination by percussion, is also of great value for indicating and estimating the kind and degree of deformity of the chest—a matter which is intimately connected with any inquiry into the cause and cure of enteroptosis. In a normal well-formed chest the sterno-ensiform line crosses the 5th costal cartilage on each side; in an emphysematous patient, or when the ribs and cartilages are abnormally horizontal in position, the line crosses the 5th space; if on the other hand the ribs are abnormally depressed, as is so common in enteroptosis, the line crosses the 4th space or even the 4th rib (Fig. 25). The sterno-ensiform line therefore serves two purposes: it gives a standard by which the degree of ptosis may be estimated, and a measure by which the deformity of the chest may be indicated.

The *transpyloric plane* or line, introduced by Dr. C. Addison, is of the greatest service for indicating the normal position of certain viscera, and

is easily applied as a matter of routine. A line drawn transversely through a point lying midway between the umbilicus and the sterno-ensiform junction—the *mid-epigastric point*—represents the transpyloric line. On each side the line cuts the costal margin near the outer border of the rectus—crossing, too, the 9th costal cartilage. Normally the pylorus is situated on the transpyloric line, about halfway between the mid-epigastric point and the right costal margin. In ptosis the pylorus is displaced downwards and towards the middle line of the body, and

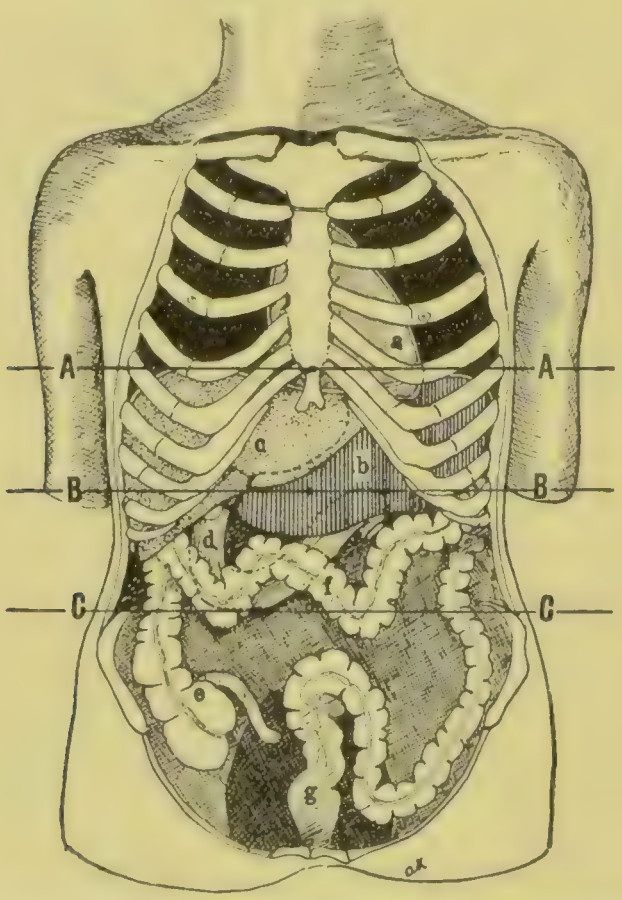


FIG. 25.—The average position of the diaphragm and abdominal viscera in healthy individuals.
A A. Sterno-ensiform plane. B B. Transpyloric plane. C C. Umbilical plane. *a*, heart; *b*, stomach; *c*, liver; *d*, duodenum; *e*, caecum and ileo-colic junction; *f*, transverse colon; *g*, rectum; *h*, gastro-hepatic omentum.

may be found at any point between the transpyloric and umbilical lines. Since the distance between these lines is usually 10 cm. (4 inches), the pylorus when displaced by a descent of the viscera is usually found between 10 and 15 cm. (4 to 6 inches) below its normal position.

The mid-epigastric point affords the most useful base for estimating the degree to which the stomach has become displaced. Dr. Addison found—and this I can confirm from considerable experience—the lesser curvature of the stomach to be situated 20 mm. (about $\frac{3}{4}$ of an inch) above the mid-epigastric point, and the greater curvature to cross the

mid-line of the body about 30 mm. (nearly 1¼ inch) below that point. In ptosis, the lesser curvature comes to lie 20 to 40 mm. below the mid-epigastric point, while the greater curvature reaches the umbilicus (10 cm. below the mid-epigastric point), or even 25 or 50 mm. below the umbilicus. Thus, in ptosis the greater curvature, instead of lying 30 mm. below the mid-epigastric point, occupies a similar position below the umbilicus, shewing a total descent of 10 cm. (4 inches). At the mid-epigastric point the lower margin of the liver crosses the mid-line of the body;

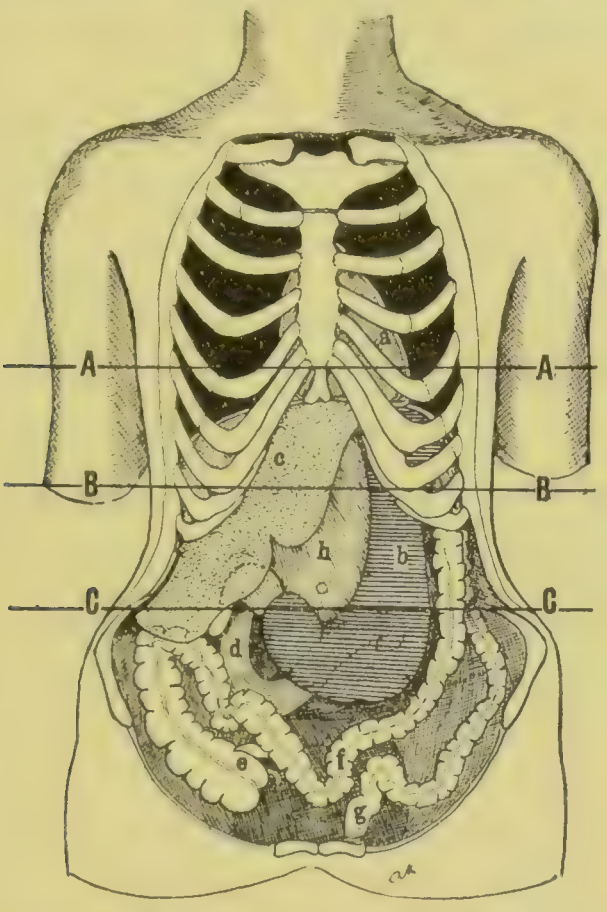


FIG. 26.—The position of the diaphragm in well-marked cases of visceroptosis. For explanation of lettering see Fig. 25.

behind it, deep in the epigastrium, the body of the pancreas crosses the vertebral column; the coeliac axis and superior mesenteric artery rise from the aorta about 30 mm. (1¼ inch) above the mid-epigastric point.

For estimating the normal position and displacements of the transverse colon and kidney, the most useful surface-marking is the umbilical line, or, if greater precision be required, the line drawn between the highest points of the right and left iliac crests. In the majority of patients the difference between the levels of these two lines is so slight that for practical purposes they may be treated as one. Normally, the transverse colon crosses the mid-line of the belly just above the umbilical

line; in ptosis it is displaced downwards 8 or 10 cm. to the level of the anterior superior spines. The lower pole of the kidney, the only part of the organ available for palpation in normal individuals, does not reach the umbilical line; the lower pole of the right kidney is situated 30 mm., and that of the left 35 mm., above the level of the iliac crests and umbilical line. On the dorsal aspect of the body the kidneys lie 1 inch (25 mm.) beyond the outer margin of the erector spinae; and anteriorly they reach just beyond the lateral margin of the rectus abdominis. In ptosis the lower pole of the kidney reaches or passes below the umbilical line, and at the same time turns inwards beneath the outer margin of the rectus.

Four other surface-markings employed in determining the displacements of the abdominal viscera must be mentioned: (1) For the lower margin of the liver.—The right subcostal margin, from the outer border of the rectus to the tip of the last palpable rib (in 30 per cent of cases the eleventh), marks the position of the lower margin of the liver when the patient is supine; in the upright posture it may descend 10 or 20 mm. below the margin. In ptosis the lower margin of the liver reaches the level of the right iliac crest, or may descend into the right iliac fossa when the upright posture is assumed. (2) For the hepatic flexure of the colon.—The right subcostal margin also marks the normal level of the hepatic flexure of the colon, which lies below and behind the part of the liver covered by the 10th and 11th costal cartilages. (3) For the splenic flexure.—This flexure ascends to a high level in the left hypochondrium, passing to a variable distance above the transpyloric line, between the greater curvature of the stomach and the spleen. In all cases of ptosis the hepatic flexure is displaced, usually reaching or passing below the umbilical line; on the other hand, the splenic flexure, like the left kidney and spleen, is rarely displaced. (4) For the caecum. —The most practical manner of indicating its position in the right iliac fossa is to place the right hand on the abdomen of the patient so that the anterior superior iliac spine lies in the fork between thumb and forefinger, the forefinger lying along the crest of the ilium. The palm then lies over the position of the caecum. The healthy caecum gives a peculiarly resonant note on percussion, and its limits can thus be defined. It is frequently displaced into the pelvis when there is no enteroptosis, and conversely in ptosis it may occupy a high place in the iliac fossa.

The extent to which the pelvic floor is depressed, and the manner of estimating the depression in enteroptosis, will be dealt with in describing the etiology of the condition.

The Means by which the Abdominal Viscera are maintained in Position.—Since visceroptosis is a prolapse or sinking of the abdominal viscera, more especially of those situated above the level of the umbilicus, it is clear that in such cases the means by which the viscera are maintained in their normal position have become disturbed and inefficient. What are these factors? At the present time three are cited, and commonly in the following order of importance: (1) Ligamentous;

(2) Atmospheric pressure; (3) Intra-abdominal pressure maintained by the muscular walls of the abdomen. Misconception as to the relative importance of these three factors is responsible for the numerous and diverse hypotheses as to the cause and nature of enteroptosis.

(1) *The ligamentous support* consists of the various folds or reflections of peritoneum binding the viscera to the abdominal wall; these folds usually contain vessels and connective tissue by which the peritoneal coverings, highly elastic and freely extensile, are strengthened. The part played by ligamentous bonds is easily shewn; if the muscular walls be stripped from the thorax and abdomen of a cadaver, which is then placed in an upright position, it will be found that in every case, unless dense pleural and peritoneal adhesions be present, the viscera drop at once into the position seen in extreme visceroptosis. In a characteristic instance, selected from numerous experiments, I found that in turning the cadaver of a young man into the upright position, the upper border of the right lobe of the liver fell to a point 65 mm. below the sterno-ensiform line, and dropped 40 mm. in the middle line of the body. Changes also occurred in the shape and the relative position of the axes of the liver, results corresponding exactly to those known to take place in enteroptosis. While the upper margin descended 65 mm. only, its lower margin descended 150 mm., because in addition to descending, the liver also became flattened from back to front, so that its vertical diameter was increased by 85 mm., and its costo-vertebral diameter correspondingly shortened. The fundus of the stomach descended 50 mm.; the pylorus fell from its normal position to the neighbourhood of the umbilicus. The lower pole of the kidneys came down to the iliac crests; the lower pole of the spleen, instead of being just below the transpyloric plane, fell downwards and forwards until it lay exposed beneath the left costal margin at the level of the umbilicus. The duodenum and head of the pancreas descended to a less extent, but the fall of the duodeno-jejunal flexure was marked (35 mm.). The transverse colon fell below the level of the anterior superior iliac spine; the caecum, the hepatic flexure, and to a less degree the splenic flexure, became prolapsed, each descending 50 mm. or more. The small intestine fell so that more than half of the small intestine lay 25 to 45 mm. below the level of Poupart's ligament and the symphysis pubis. On reversing the subject and placing the head downwards, practically all the organs of digestion entered the hypochondria, all the viscera except some parts of the colon passing beyond the level of the umbilicus. The ligaments were sufficiently loose to allow the upper limits of the liver and stomach to move from a point 65 mm. below the sterno-ensiform line to one 35 mm. above it—a total movement, due to inversion of the body, of 100 mm. (4 inches). With the inversion of the body the kidneys shew a total movement of 35 to 70 mm. ($1\frac{1}{2}$ to $2\frac{3}{4}$ inches). That such a mobility is not a post-mortem effect may be proved by the free respiratory movements of patients with the abdominal type of respiration. In such individuals the right dome of the diaphragm can be

seen to have an excursion in extreme respiratory efforts of 60 to 80 mm. Those who regard the visceral ligaments as the chief means of support, and the majority of writers on enteroptosis have held this opinion, forget that all the viscera situated within the supra-umbilical or respiratory zone of the abdomen must be loosely held to allow the visceral movements caused by respiration. They also overlook the very important point that were the weight or part of the weight of the viscera thrown on these peritoneal bonds, such a traction or tension must strain the various vessels and nerves contained within them and interfere with their function. The result of such traction is seen in cases of enteroptosis when the normal support has become deficient.

(2) *Atmospheric Pressure.*—In most of the medical schools of Great Britain it is still taught that atmospheric pressure assists in maintaining the liver within the right dome of the diaphragm. That atmospheric pressure has absolutely no part in the normal maintenance of the liver may be proved thus: (*a*) When a cadaver with intact body-walls is set upright, the liver becomes partly prolapsed beneath the costal margin; the epigastrium is drawn in to occupy the space vacated by the liver. (*b*) There is always a positive pressure in the uncontracted stomach, which occupies the left dome of the diaphragm. Moritz and Schwerdt, Kelling, and the writer independently of these three observers, estimate that the intragastric pressure when the stomach is passive is 4 to 8 mm. Hg when standing upright during expiration, and rises to 12 mm. Hg during inspiration—that is to say, the diaphragm is always in a state of tone or contraction, exerting pressure on the liver, stomach, and abdominal contents. Schreiber, on the other hand, found a negative intragastric pressure of ·5 to 1·5 mm. Hg. By taking a deep thoracic breath, and holding the abdominal walls as rigid as possible, I found it possible to produce a negative intragastric pressure of − 4 mm. Hg This is indicated also by the retraction of the epigastrium. The intragastric pressure in enteroptosis has not, as far as I know, been investigated, but there can be little doubt that, in the standing posture, a negative pressure will be found, at least in the early stages; for the epigastrium becomes drawn inwards in such cases to fill up the vacuum. It is just possible that Schreiber's observation was made on an individual with a tendency to ptosis. In cases of enteroptosis Schwerdt found that the intra-abdominal pressure, as estimated per rectum, was lowered.

(3) *The Viscera are normally supported by the indirect action of the Muscular Parietes of the Abdomen.*—In medical literature very little is to be found relating to the intra-abdominal pressure or tension, which is caused and maintained by the tonus and contraction of the abdominal musculature. Yet this force keeps the viscera in position, and regulates, and to a considerable extent maintains, the venous circulation of the abdomen. On placing a cadaver in the upright position, ptosis of the viscera occurs; but when a living healthy man rises from the prone to the erect posture, the muscular walls of the abdomen come reflexly into action; the domes of the diaphragm in a few cases may not sink at all,

there being no descent of the viscera; in the majority of cases examined, however, the domes fall from 10 to 15 mm., and the lower margin of the pylorus descends to a corresponding extent. In the erect posture there is a positive pressure of 20 to 28 mm. in the rectum, and in the stomach, as has just been pointed out, of 6 to 12 mm. Hg. The increase of pressure in the rectum, over and above that in the stomach, is undoubtedly due, as Schwerdt points out, to the weight of the superincumbent viscera. The pressure exerted on the abdominal contents by the abdominal muscles, maintaining one viscus against another, may therefore be that estimated at 6 to 12 mm. Hg; but when the muscles of the abdomen are thrown into action it may temporarily be higher than the arterial blood-pressure. On stooping down and then lifting a heavy weight, the pressure within the stomach rises to over 70 mm., that within the rectum to 120 mm. Hg; in coughing, in straining, and bending temporary elevations of 50 or 90 mm. Hg are observed. In strong, muscular men the intra-abdominal pressure may rise much higher—possibly to 200 mm. Hg.

The muscles of the abdomen do not take an equal part in maintaining the viscera in position. In the upright posture the diaphragm works with gravity; in the supine position against it; but in the horizontal, as in the upright posture, its action is to depress the viscera. The share taken by the levator ani is mainly passive. The muscles which maintain and regulate the intra-abdominal pressure are the transversalis, internal and external obliques; the recti abdominis are rather concerned in the movements of the body than in maintaining the intra-abdominal pressure and thus supporting the viscera. Now the action of the transversalis and internal and external oblique muscles on the viscera is especially well seen during a forcible expiratory movement such as coughing. The viscera situated above the umbilicus are forced upwards into the hypochondria, and by displacing the diaphragm upwards compress the thoracic viscera. The infra-umbilical viscera are forced downwards against the hypogastric region of the abdominal wall and into the pelvis. In brief, the effect of contraction of the transversalis, internal and external oblique muscles is to press the supra-umbilical viscera upwards and the infra-umbilical viscera downwards. They are the only muscles of the abdominal wall which normally support the supra-umbilical viscera. To obtain a true conception of enteroptosis it is of the first importance to recognise these two regions of the abdomen—the supra-umbilical and infra-umbilical. The supra-umbilical region contains the viscera which become prolapsed in enteroptosis — the liver, stomach, kidneys, transverse colon, duodenum, pancreas, and spleen. The muscles in the supra-umbilical part of the abdominal wall take an active part in respiration; in abdominal respiration they are the active opponents of the diaphragm, and by pushing up the supra-umbilical viscera they elongate its fibres; in thoracic respiration, on the other hand, they stay or brace these viscera which then form a base or fulcrum used by the diaphragm to elevate the ribs and thorax during inspiration.

The infra-umbilical part of the abdominal wall takes a passive part only in respiration; its muscles support the infra-umbilical viscera, which in their turn form a shelf for the supra-umbilical contents of the diaphragm. When the muscles—the two oblique and transversalis muscles—which support the viscera contract, as in expiration, the viscera of the upper region ascend, and those of the lower region descend. If a viscus drop from the upper to the lower region the direction of its respiratory movement is then reversed.

Changes in the Shape, Position, and Fixation of Viscera with the onset of Visceroptosis.—In the upright posture the viscera are so supported that no strain or weight falls on their ligamentous and vascular supports. But when from any cause the muscular support is partly or completely withdrawn, changes occur in the shape and position of the viscera and a strain falls on their ligamentous supports. The changes which ensue in each organ depend on its means of fixation, and it is therefore necessary to give a brief account of the ligamentous bonds in connexion with each abdominal viscus.

Ligamentous and Vascular Bonds of the Liver.—In the upright posture the lower or visceral surface of the liver rests on a shelf formed by the right kidney, colon—especially its hepatic flexure, stomach, first stage of the duodenum, gastro-hepatic omentum, pancreas, and coeliac axis. The visceral shelf is maintained by the contraction of the abdominal wall. The upper or parietal surface of the liver is pressed against the diaphragm and a small area of the anterior abdominal wall in the epigastric region. Only behind is the liver really fixed, being attached to the diaphragm by (1) hepatic veins and inferior vena cava, (2) connective tissue, and (3) a reflection of peritoneum; this triple bond forms the *mesohepar*. The falciform and round ligaments are so loose and so elastic that the freest movement is allowed to all parts of the liver, except the posterior, which is attached by the mesohepar. Now, if the visceral shelf be withdrawn by relaxation of the abdominal wall, the liver, being hinged behind by the mesohepar, falls downwards and backwards, undergoing at the same time a rotation on its mesohepatic hinge. The liver becomes flattened from back to front, its lower border is greatly depressed; a strain falls on the mesohepar. A similar change may be produced in the liver by quite another process; if the space in which it is situated be reduced in size either by compressing the thorax with corsets or by an exaggeration in the action of the external oblique muscle, the most movable part of the liver—the anterior part—is extended, the liver at the same time undergoing the same rotation as occurs in true ptosis. The result is the same in both cases, but the forces at work are very different.

The strain which falls on the hepatic artery and the structures which lie with it in the gastro-hepatic omentum and transverse fissure of the liver is important. Normally, the transverse fissure is on a level with the coeliac axis, from which the hepatic artery arises. In enteroptosis the transverse fissure descends as much as 50 mm. or more below the

level of the coeliac axis, some degree of strain thus falling on the hepatic artery and on all the structures in the transverse fissure of the liver.

Ligamentous and Vascular Supports connected with the Stomach.—At two places only is the stomach firmly attached, its cardiac orifice is bound to the diaphragm and tissues of the posterior mediastinum by the oesophagus and the reflections of peritoneum and connective tissue surrounding the oesophagus. The pyloric end, which has a normal up-and-down and side-to-side movement of 30 to 50 mm. (1¼ to 2 inches), is fixed by the gastro-hepatic omentum to the transverse fissure of the liver, and by the hepatic artery, the pancreas, and the connective tissue surrounding these two structures, to the aorta, the aortic orifice of the diaphragm, and the solar plexus.

Ligamentous Bonds of the Duodenum and Pancreas.—The body of the pancreas crosses the aorta in the narrow fork between the coeliac axis above and the superior mesenteric artery below, and is bound by fibrous tissue to the aorta and the aortic orifice of the diaphragm. But although the most firmly attached of all the viscera, the pancreas may be displaced downwards to the extent of 50 mm. (2 inches) in enteroptosis. In gastroptosis this part of the pancreas is commonly palpable in the epigastrium above the lesser curvature. The duodenum and the head of the pancreas within its bend are pushed downwards by the falling liver, the lower bend of the duodenum crossing the vertebral column near the promontory of the sacrum. In this condition, too, the duodenum is dilated, hypertrophied, and frequently presents the diverticula described by Dr. Rolleston (47). (*Vide* p. 567.)

Ligamentous Bonds of the Spleen.—The spleen rests against the anterior and upper part of the left kidney. It is loosely bound to allow of free movement during respiration and filling of the stomach. Above it is bound to the diaphragm by the suspensory reflection of peritoneum, below also to the diaphragm by the left costo-colic ligament on which the lower pole rests; between these attachments it is connected to the capsule of the left kidney by the lieno-renal ligament which may be either a narrow mesentery or a wide and extensive adhesion. In only 2 per cent of cases of enteroptosis is the spleen markedly displaced; this is not because the spleen is firmly fixed, but because it is relieved and saved from displacement by the free mobility of the stomach, gastric contents, and transverse colon.

Ligamentous Bonds of the Small Intestines.—The small intestines, supported by the infra-umbilical part of the abdominal wall, form the chief part of the visceral shelf on which the supra-umbilical viscera rest. When the hypogastric wall or pelvic floor gives way the small intestines sink down and strain thus falls on the mesentery. The chief supporting structures of the mesentery are the large superior mesenteric artery between its layers and the fibrous sheath surrounding the artery. The fibrous sheath commences in the firm, strong tissue round the coeliac axis and aortic orifice of the diaphragm. The reflections of peritoneum at the root of the mesentery are lax and freely movable. In enteroptosis the

superior mesenteric artery and the nerve plexuses on it are elongated and the reflections of peritoneum at the root are displaced downwards, 2 inches or more.

Ligamentous Bonds of the Large Intestines.—At birth the position of the caecum is more variable than that of any abdominal organ. It may lie in front of the left kidney, or just below that organ or lower still in the iliac fossa. It is not attached directly to the abdominal wall, but is supported indirectly by the termination of the ileum and ascending colon. It is freely movable, and in rather more than half the cases of enteroptosis is displaced into the pelvis.

The hepatic flexure is bound in front of the right kidney to the perirenal capsule and to the duodenum. A fold of peritoneum—the right costo-colic fold—gives it a loose attachment to the right wall of the abdomen below the liver. Traction on the hepatic flexure pulls forwards the capsule of the kidney and the kidney within it. When the right lobe of the liver becomes prolapsed, the hepatic flexure is pushed downwards. Displacement of the kidney may not affect the attachment of the hepatic flexure. The transverse colon is the most loosely bound organ in the abdomen. It is attached across the posterior abdominal wall by the mesocolon, and to the stomach by the great omentum.

Ligamentous Bonds connected with the Kidneys.—The kidneys are freely movable within the fat-lined perirenal capsules which lie behind the peritoneal cavity. They are so closely in contact with the diaphragm that they participate in all the respiratory movements of that muscle. The upward and outward movements of the kidneys are limited by the union of the anterior and posterior layers of the perirenal capsule with the firm subperitoneal tissue on the diaphragm and transversalis muscle. Over the lower pole of the kidney these two layers remain open and descend on the ureter and allow a free descent of the kidney. In enteroptosis there is really a double displacement: first, a downward and inward displacement of the kidney within its perirenal capsule, the upper empty part of which is then seen to form a fibrous stratum passing up to the suprarenal body; secondly, a displacement downwards of the whole perirenal capsule and kidney-bed. The only bond which fixes the kidney directly to the abdominal wall is formed by the vessels, nerves, and fibrous tissue passing to the hilum. This bond forms the tether round which the kidney rotates in its descent; as it descends the lower pole necessarily passes inwards and forwards, and may come forwards either above or below the transverse colon. In either case it also pushes the duodenum and head of the pancreas in front of it. Although the perirenal capsules and ligamentous bonds of the two kidneys are equally firm and strong, displacement of the right kidney is ten times more frequent than of the left. The spleen, the splenic flexure of the colon, and the left kidney are safeguarded by the ease with which the stomach and transverse colon drop from the left hypochondrium.

Ligamentous Bonds of the Pelvic Viscera.—In enteroptosis the pelvic

viscera are displaced, but to a variable extent. They are supported (1) by the levator ani; (2) by strong ligamentous bonds which bind them to the symphysis pubis and white line of the pelvis. The pelvic peritoneum is so loosely attached and so movable that it gives practically no support to the pelvic viscera.

Degrees and Forms of Visceroptosis.—Although Glénard defined enteroptosis as a prolapse or sinking of the abdominal viscera, his diagnosis of the condition rested on the recognition of four minor points: (1) on the condition of the colon; (2) on the position and mobility of the kidneys (especially the right); (3) on the position of the pyloric part of the stomach; (4) on the position of the lower border of the liver. If the colon could be felt narrowed and cord-like, the kidney palpable during inspiration by his special mode of examination, the stomach felt or recognised at the level of the umbilicus, the lower border of the liver 4 to 6 cm. (2 to 3 finger-breadths) below the costal margin, he diagnosed enteroptosis. Even when two only of these physical signs were present he made the diagnosis of enteroptosis, provided the symptoms and history answered to his clinical picture of the condition. But, as will be seen presently, numerous patients present all Glénard's four signs without there being any total prolapse of the viscera. After it was shewn in my Hunterian lectures on enteroptosis (27) that in all cases of true ptosis or sinking of the viscera the diaphragm occupied a very low level (complete expiratory position), and that its muscular fibres were reduced to half their normal length, Glénard (19) proposed that such cases should be classed under the term "Phrenoptosis." The proposal is unfortunate, for there can be no phrenoptosis unless true enteroptosis be present. The numerous cases published by Glénard may be divided into at least two distinct classes: (1) in which the diaphragm has sunk into an expiratory position, and there has been a complete subsidence of the supra-umbilical viscera; (2) in which the domes of the diaphragm occupy a normal position but the lower limits of the viscera in the supra-umbilical region of the abdomen occupy an abnormally low position. The cases belonging to Class I. shew true or complete ptosis; those of Class II. false or partial ptosis. The cases of partial ptosis are again divisible into two distinct groups: (*a*) due to enlargement of the hypochondriac viscera; (*b*) resulting from compression of the hypochondriac spaces. The normal bulk of the liver varies from 1500 to 1800 c.c., yet under a low pressure 1000 to 1200 c.c. of fluid can be injected into it by the portal vein; if the hypochondriac space remain of the same size the injection causes the lower border of the liver and the viscera under the liver to descend from 25 to 35 mm. (1 to 1½ inch), a fluctuation frequently seen in the course of an illness. Enlargement of the stomach or spleen gives a similar result. This form of ptosis may be distinguished as the partial *ptosis of visceral enlargement.* Compression of the hypochondriac spaces is due to the use of corsets, to depression of the ribs by over-action of the muscles of expiration, or other causes. In partial *ptosis due to compression* there is, besides a prolapse of the supra-

umbilical viscera, also a depression or ptosis of the infra-umbilical organs due to the compression of the waist.

The classification proposed is the following: (1) *Complete enteroptosis.* There is a prolapse of both supra- and infra-umbilical viscera. (2) *Partial enteroptosis due to visceral enlargement.* Only the supra-umbilical viscera may be affected. (3) *Partial enteroptosis caused by compression.* Both supra- and infra-umbilical viscera are always affected.

The Incidence of Visceroptosis as regards Age and Sex.—In 110 subjects, 40 examined by Dr. Addison and 70 by myself, there were 14 (6 males, 8 females) which presented all the features of complete enteroptosis. All the 14 shewed gastroptosis, prolapse of the liver, descent of the duodenum, of the transverse colon, and hepatic flexure. In all but one the right kidney reached or passed beyond the iliac crest; in only one was the left kidney low; in seven the caecum lay within the pelvis; in three it was partly within the pelvis; in four it was in the iliac fossa. These subjects were obtained chiefly from a workhouse population, and half of them were over 55 years of age. There were also 23 cases of partial ptosis, 10 being males and 13 females. Thus, among 58 male subjects 16 (27 per cent) presented partial or complete enteroptosis; and of 52 women 21 (40 per cent) presented a similar condition. In a series of 1000 patients (519 males, 481 females) Glénard (18) found partial or complete ptosis in 30 per cent of the men and 40 per cent of the women—practically the same percentage as is found in subjects drawn from the workhouse population of England. Rose examined 100 consecutive patients in Einhorn's clinic in New York; in 16 he found the stomach below the umbilicus; only nine of these complained of gastric symptoms. Koellreutter noted that 10 per cent of the women attending a dispensary in Heidelberg shewed symptoms and signs of complete enteroptosis. He records 50 cases in women: the visceral displacements correspond exactly to those we found in English post-mortem subjects. Einhorn estimates its occurrence at 25 per cent amongst women. The condition is rare in children. Rose gives an account of the condition and treatment of such cases in childhood; Phillips records a case of a new-born child with floating kidneys. Most women who come under treatment for enteroptosis are between the ages of 25 and 40 years.

The Influence of Age on the Position of the Viscera.—There takes place during the lifetime of every one a certain degree of splanchnoptosis, due (1) to the alteration in the shape of the thorax and of the angle at which the lower ribs are set: (2) to a loss of elasticity and tone in the tissues of the body-wall and in the lungs. Between the 25th and 70th year the lower border of the lung descends nearly a rib's breadth (Feitelberg, C. Schmidt): the anterior ends of the ribs are depressed from 10 to 20 mm., so that the costal position becomes more vertical; the antero-posterior diameter of the chest decreases; except in emphysema the hypochondriac spaces decrease in capacity; the oesophagus elongates 20 to 30 mm. on account of the descent of the diaphragm (Kolster), and the larynx descends 30 to 40 mm. (Mehnert).

Etiology.—The explanations given of the origin and nature of enteroptosis are extremely numerous and diverse. It is generally thought that the immediate cause of complete enteroptosis is a fall of the intra-abdominal pressure. The reduction, in Glénard's opinion, is chiefly due to a diminution in the calibre and contents of the intestine—especially of the colon; many writers refer it to the sudden reduction of the abdominal contents which follows parturition, especially when the child is large; others consider that it results from a rapid emaciation and absorption of fat. As diminution of the intra-abdominal pressure may also be produced by an enlargement or relaxation of the abdominal walls, it has been thought that enteroptosis is due to a disturbance of the innervation of the abdominal muscles (Schwerdt), to their atony or weakness, to separation of the recti abdominis, to stretching of the perineal floor, to the presence of a hernia, or a congenital defect in the structure of the abdominal wall. On the other hand, it has been maintained that enteroptosis is the result, not of a decrease, but of a sudden increase of the intra-abdominal pressure, such as occurs during straining at stool (Arbuthnot Lane), coughing, and similar acts. Constriction of the waist by corsets or belts has often been regarded as a direct cause (Chapotôt, Hayem, Terrier and Auvray), as have high heels, fashionable exaggerations of the lumbar curvature, deformities of the spine and of the thorax. Congenital elongation of the visceral ligaments is held by many as a cause, but there is not the slightest evidence that such a condition ever occurs; a floating 10th cartilage or free 10th cartilage, which occurs in 50 per cent of individuals, has been regarded as a constant accompaniment of enteroptosis (Stiller); chlorosis, neurasthenia, nervous dyspepsia, dyspepsia, disordered action of the liver, venous engorgement within the abdomen, pulmonary tuberculosis, rickets, and the nervous exhaustion and muscular weakness following infectious diseases have all been incriminated. When so many explanations are offered by competent authorities the presumption is justified that the true nature and real cause of enteroptosis are not rightly understood. One of the reasons for the confusion is the failure to recognise that three distinct conditions are included under the one term; namely, complete or true enteroptosis, partial enteroptosis due to visceral enlargement, and partial enteroptosis due to compression. It is with the etiology of the complete form that we are here concerned.

Every one who has had practical experience of patients suffering from enteroptosis agrees that the application of a belt or of any form of abdominal support gives immediate amelioration or complete relief. An inquiry into the manner in which such supports act will throw light, not only on the functional disturbance associated with enteroptosis, but also on the production of the ptosis. We have seen that in enteroptosis the weight of the viscera falls to an undue extent on their ligamentous and peritoneal supports, and that a strain is thrown on the vessels lying in those supports. Application of a belt will support the viscera and relieve the strain. But the action of a belt is probably much more than this.

Dr. Leonard Hill has demonstrated that a compensatory mechanism exists within the abdomen for regulating the blood-pressure when the upright posture is assumed; if this mechanism be imperfect, the blood collects within the venous system of the abdomen and a temporary cerebral anaemia or syncope is produced. As Dr. Hill expresses it, patients with an imperfect compensatory mechanism bleed into their own abdomens in the upright posture. In this mechanism the muscular walls of the abdomen play an important part; by their contraction they can force the blood on to the heart. Dr. Hill shewed that a similar effect could be produced by the application of an abdominal belt. Hertzka and Buxbaum inferred that venous engorgement exists in enteroptosis; in short, all the symptoms point to a disturbance of the splanchnic compensatory mechanism. The vasomotor disturbance, shewn by morbid flushing, dizziness, the faintness, palpitation, dyspnoea, and exhaustion on making the slightest exertion, all point to the same condition, namely, stagnation of blood within the abdomen, which is dispelled by the application of abdominal pressure or the recumbent posture. Enlargement of the superficial abdominal veins, regarded by Ott as one of the signs of enteroptosis, indicates plethora of the great systemic and portal veins of the abdomen. The application of an abdominal support immediately eases the movements of respiration, slows and improves the heart-beat, and raises the arterial blood-pressure. The same effect is obtained by placing the patient in the recumbent position. Subjects of enteroptosis, according to Glénard, find they can obtain sleep, when other means fail, by compressing the abdomen, by lying face downwards, or by applying direct pressure. All these results point clearly to a disturbance of the circulatory mechanism within the abdomen which results in a stagnation or collection of the venous blood within that cavity.

Besides relieving the strain on the ligamentous supports of the viscera and restoring the venous circulation in the abdomen, the application of a belt to the subjects of enteroptosis has a very direct effect on the action of the diaphragm, which was first shewn to me by Professor Wenckebach of Groningen. In such patients the diaphragm has permanently assumed a contracted or inspiratory position, its movements being curtailed or almost absent. But directly a support is applied to the lower part of the abdomen, the epigastrium fills out and, by the aid of the radioscopic screen, the diaphragm is seen to be pressed up, assuming an expiratory position; at the same time its movements again begin, or if previously restricted, shew increased amplitude. Glénard (19) found that the diaphragm was forced upwards, in such cases from 20 to 30 mm. (1 to $1\frac{1}{4}$ inch), but in old-standing cases, as Wenckebach demonstrated, the contraction of the diaphragm had become almost permanent and difficult to alter. The diaphragm in these cases has been thrown out of action because its opponents—the muscles of the anterior abdominal wall—have become incapable of their full work. A muscle may be thrown out of action just as completely by paralysing its normal

opponent as by intrinsic paralysis. A belt, then, restores the action of the diaphragm by restoring or taking the place of its normal opponent. Pregnancy produces the same effect in another way ; it is well known that the protean symptoms of enteroptosis disappear during pregnancy, only to reappear in an exaggerated degree at its close. There is no doubt that the disturbed or restricted action of the diaphragm is one of the main features of enteroptosis. The action of the diaphragm is as closely connected with the circulation of blood within the abdomen as with air within the lungs (28). Wenckebach's clinical observations emphasise the circulatory importance of the diaphragm and abdominal walls, thus agreeing with my deductions from comparative morphology. In enteroptosis the circulatory function of the abdominal wall is profoundly disturbed. How is this disturbance caused ? François-Franck found that placing a four-footed animal in the upright position produces enteroptosis ; the viscera gradually subside, bulging out the lower part of the abdomen, causing a sinking in or narrowing of the upper part ; the diaphragm follows the viscera and passes into the inspiratory position, its contractions being wholly exerted on its costal attachments which are drawn inwards. Respiratory movements are confined to the upper part of the thorax as in cases of enteroptosis. While the costal part of the diaphragm is thrown out of action, the spinal part still remains in action. This I have observed in early cases of enteroptosis. If chloroform be given to an animal placed in the upright position, profound enteroptosis appears almost immediately, and the animal dies from syncope ; chloroform, then, destroys at once the compensatory mechanism. The tonus and contraction of the musculature of the abdominal wall are influenced reflexly by the state of the abdominal viscera. Dr. J. Mackenzie has brought forward much clinical evidence in favour of the existence of a viscero-muscular reflex or mechanism ; Dr. Head's observations leave no doubt as to the intimate manner in which visceral states are reflected on the abdominal wall. Disordered conditions of the liver, stomach, duodenum, kidney, and appendix give rise to contractions—often sharply localised—of the abdominal musculature. The afferent impulses reach the spinal cord through the great splanchnic, and leave by the intercostal nerves for the abdominal wall. When the upright posture is assumed the abdominal muscles respond reflexly to the increased burden thrown on to them by the viscera. The mesenteries contain numerous Pacinian corpuscles, generally regarded as end-organs for receiving pressure-stimuli which, on reaching the central nervous system, regulate and co-ordinate the muscles of the part whence the stimuli have come. Experimental evidence is still wanting to shew that the abdominal musculature is so related to the Pacinian corpuscles of the mesenteries. Schwerdt has shewn that the intra-abdominal pressure is lower than normal in the subjects of enteroptosis. When a healthy man is placed in the Trendelenburg position the pressure within the rectum is still positive ; but in women, especially in those with enteroptosis, a considerable negative pressure appears. Even in normal individuals, anaesthetised in the Trendelenburg

position, there is a negative pressure in the raised part of the abdomen. When the subjects of enteroptosis are placed in a similar position, Schwerdt found that a negative pressure, varying from − 4 to − 7 mm. Hg., appeared in the rectum ; in the same position normal individuals shew a positive pressure of 2 to + 4 mm. Hg. Unfortunately, nothing is known as to the intragastric pressure in such subjects, but the sinking in of the epigastrium, on assuming the upright posture, shews that it is a negative instead of a positive pressure. (*Vide* p. 866.)

The abdominal walls are flaccid and thin in the subjects of ptosis ; the thickness of the muscular wall, measured between the crest of the ilium and the costal margin, varies from 5 to 15 mm. ; in normal subjects it varies from 7 to 25 mm. Koellreutter estimated that 30 to 50 per cent of the cases of enteroptosis in women are the direct result of pregnancy ; Glénard's estimate is 60 per cent. According to Glénard, the muscular fibres of the internal oblique and transversalis which are within the supporting or infra-umbilical region of the belly wall, are not increased in length during pregnancy. The increase in the circumference of the abdomen is wholly due to a widening of the linea alba, but in this expansion the lower 70 or 80 mm. (3 inches) of the white line should take no part. If it does, separation of the recti occurs, resulting in flaccidity of the supporting or hypogastric part of the abdominal wall and ptosis of the viscera. In enteroptosis there is an atonic condition of the visceral musculature. Mall has shewn that peristaltic movements drive the portal blood onwards ; in enteroptosis this force is certainly diminished, so that, from this cause also, some degree of venous stagnation results. In persons with relaxed abdominal walls the liver is soft and flaccid, a sign of low blood-pressure in the portal area. All these circumstances point to a close connexion between the portal circulation and the tone of the abdominal walls, and also to the possibility that portal stasis, caused by a disordered liver, disturbs the viscero-muscular reflex mechanism regulating the tone of the abdominal wall.

Summary of the Etiology of Complete Visceroptosis.—Complete enteroptosis is accompanied by a relaxation of the abdominal wall and venous stasis within the abdomen. The visceral ptosis is due to the relaxation of the abdominal wall ; the relaxation may follow from (1) expansion of the linea alba, (2) loss of reflex tone of the muscular fibres. It is possible that the viscero-muscular reflex which regulates the tone may be upset by a portal stasis following functional disorders of the liver.

Etiology of Partial Ptosis.—Tight compression of the waist by belts or corsets produces the condition of *partial ptosis from compression.* When the waist is at the level of the umbilicus the application of corsets forces the supra-umbilical viscera upwards and the infra-umbilical viscera downwards. Further, corsets necessarily diminish the hypochondriac spaces, and thereby dislodge part of their normal contents. The costal margin and ribs are pushed downwards and inwards ; the subcostal angle is narrowed. From each space the most movable organ or part of an organ is extruded. On the left side it is the transverse colon—a special loop

of which moves into the space when the stomach is empty—and the pyloric division of the stomach, which becomes elongated in shape and vertical in position. The spleen and splenic flexure of the colon are rarely expelled, not because they are tightly bound, but because they are saved by the free mobility of the stomach and transverse colon. On the right side the lowest part of the liver is extruded; namely, that lying under the 10th and 11th costal cartilages. When the waist is above the level of the umbilicus, crossing the 10th, 11th, and 12th ribs, the kidney may be extruded, or both the kidney and the liver. Among 1000 consecutive patients Glénard found both the right kidney and the lower part of the right lobe of the liver extruded in 11 men and 69 women. The right kidney alone in 6 men and 84 women.

The hypochondriac spaces may be diminished by other means besides corsets; in the long funnel-shaped chests of those with the *Habitus phthisicus*, the ribs are long, and their anterior ends depressed. A rib when depressed must also encroach on the hypochondriac space because of the nature of its articulation to the spine. Over-action of the expiratory muscles also diminishes the hypochondriac spaces.

Thoracic conditions can give rise to complete and true enteroptosis. Thus, Dr. Addison found it in two cases, one with pneumothorax, the other with a thoracic growth.

The muscles of the pelvic floor play a secondary part only in the production of enteroptosis. I found that in complete cases the pelvis, when the small intestine was removed from it, held from 350 to 600 c.c. of water, and that this was also true in cases of infra-umbilical ptosis.

Functional Disturbances Resulting from Visceroptosis.—(1) There is a profound disturbance of the respiratory mechanism, the lower ribs and epigastrium being rigid, or even drawn in during inspiration, and the respiratory movements mainly confined to the upper part of the thorax. If the diaphragm takes part, it is merely to help in raising the thorax. In some cases, while the supra-umbilical part of the abdominal wall is rigid, the infra-umbilical part may share in the respiratory movements as an opponent to the diaphragm. (2) The disturbance of the circulation has been described under Etiology. (3) With the prolapse of the liver the gall-bladder, instead of being at an angle of 45° to the upright axis of the body, becomes vertical in position with the fundus downwards. Prolapse of the duodenum brings some degree of strain to bear on the common bile-duct, and since the common hepatic and cystic ducts in the transverse fissure are fixed to the diaphragm by fibrous tissue at the posterior part of the longitudinal fissure of the lines, the cystic duct is sharply kinked and the common bile-duct partially obstructed by the strain. Dutton Steele found by experiment that the resistance to fluid passing from the gall-bladder into the duodenum was equal to a pressure of 15 cm. of water when the viscera were not displaced; while in ptosis the pressure required varied from 35 to 40 cm. (4) Since the diaphragmatic movements not only assist the circulation of blood in the liver but also the expulsion of bile,

the exit of bile from the liver is retarded. (5) In all cases of enteroptosis the duodenum is dilated and hypertrophied. Since ptosis of the small intestine into the pelvis causes a strain on the root of the superior mesenteric artery which passes over the third stage of the duodenum, obstruction and dilatation of this part of the intestine may thus be produced. (6) The statement is often made, but without any post-mortem evidence, that obstruction may be due to kinking at the pyloro-duodenal junction, at the hepatic flexure of the colon, or at the splenic flexure. (7) It is also said that a floating kidney may press against, and cause obstruction of, the common bile-duct. But in a case in which the right kidney had displaced the duodenum forwards and lay behind it, I could not find any evidence that any such obstruction had occurred.

Associated Conditions.—1. Neurasthenia, in 11·5 per cent of cases (Glénard), in 90 per cent (McCallum). 2. Pulmonary tuberculosis in 50 per cent (McCallum); 3. Chlorosis in 8 per cent (Koellreutter); 4. Dyspepsia, 20 per cent (Glénard), 54 per cent (Koellreutter); 5. Mucous entero-colitis; 6. Diabetes (Glénard); 7. Alcoholism (Glénard); 8. Cholelithiasis, 10 per cent (Dutton Steele); 9. Malaria (Glénard); 10. Renal lithiasis.

Symptoms and Signs.—Enteroptosis is usually discovered accidentally during the routine examination of patients coming under treatment for dyspepsia, neurasthenia, malnutrition, vague bodily pains, or unnatural fatigue on exertion. It is said to occur without giving rise to any functional disturbance.

Symptoms. — (*a*) *The circulatory disturbances* are dizziness, fainting, flushings of the head, palpitation and exhaustion on assuming the erect posture, and are relieved on lying down. (*b*) *Gastric disturbances* comprise loss of appetite, feeling of fulness after meals, nausea, vomiting, eructations, and burning sensations. (*c*) *Nervous disturbances.*—Pains or dragging sensations in the back and loins; often tenderness on palpation between the costal margins and the umbilicus; neuralgic pains in the head, sleeplessness, reflex cough, hysteria, complete loss of mental buoyancy. (*d*) *Intestinal disturbances.*—Constipation is common (70 per cent, Koellreutter); diarrhoea from mucous colitis may occur. (*e*) Sharp exacerbations of pain—Dietl's crisis—were found by Dutton Steele in 84 per cent of cases of hepatoptosis, which is invariably accompanied by enteroptosis.

Physical Signs. — (*a*) Gastroptosis is present, the stomach being vertical in position, elongated in shape, and reaching down to the umbilicus or even 7·5 cm. (3 inches) beyond it. There is: (*b*) hepatoptosis, the liver is deformed, more movable than normal, its right lobe being prolapsed into the right loin, under the 10th and 11th costal cartilages, or the whole of the lower border of both the right and left lobes may be depressed 3, 6, or 10 cm. (1 to 4 inches) below the costal margin. The consistence of the liver varies, but usually it is flaccid. Glénard employs a special method (*procédé du pouce*) to determine the position of the lower border of the liver, using the thumb of the left

hand for examining it during forced respiration. The right flank of the patient is grasped by the left hand, so that the fingers pass into the loin behind and the thumb into the abdomen in front, some distance below the costal margin. At the same time the fingers and palm of the right hand are applied firmly to the abdominal wall between the umbilicus and the right anterior superior iliac spine in order to press the viscera upwards against the under surface of the liver, so that the lower border of that organ, as it descends during inspiration, necessarily rides forwards against the wall of the abdomen, and is felt by the thumb of the left hand. (*c*) Nephroptosis on the right side occurs in all cases of complete ptosis. At the end of inspiration, the right kidney can be felt and grasped in the right flank. (*d*) A linguiform process of the liver (Riedel's lobe) or the gall-bladder may also be discovered in the right loin, with or without a displaced kidney, in partial ptosis. (*e*) The spleen, left kidney, and splenic flexure of the colon are found displaced and palpable in about 2 per cent of cases only. (*f*) The pancreas can commonly be felt by deep palpation in the epigastrium, midway between the umbilicus and the sterno-ensiform point. It lies above the lesser curvature of the prolapsed stomach ; it may be covered by descent of the left lobe of the liver. (*g*) The transverse colon may be recognised by its contracted condition. It is freely movable upwards and downwards, but not from side to side (Glénard). (*h*) The caecum may also be palpable on account of its contracted condition ; in advanced cases it becomes dilated. It is displaced into the pelvis in about 50 per cent of the cases. (*i*) The vagina is unduly pressed downwards, so that its axis is directed towards the tip of the coccyx, and its roof is depressed by the bladder. The uterus is retroflexed, and its fundus pressed into Douglas's pouch. (*j*) The abdominal wall is unusually flaccid and the viscera unduly movable. In early stages of the disease the supra-umbilical part of the abdominal wall, especially the recti muscles, is rigid. Areas of tenderness are often present, and palpation causes a reflex tension of the upper abdominal musculature. A line drawn by the nail on the skin leaves a red streak, shewing a vasomotor disturbance of the cutaneous vessels. (*k*) When the patient stands up the supra-umbilical part of the abdominal wall sinks in and the infra-umbilical part bulges out. The lower border of the liver may descend 2 or 3 inches. In passing from the recumbent to the standing posture, the distance between the anterior superior spines will increase by 30 per cent or more, while in a normal individual it increases 25 per cent or less. (*l*) From sinking in of the epigastrium and the prolapse of the stomach, the abdominal aorta, and the coeliac axis are palpable. Pulsation is commonly seen in the epigastrium. (*m*) Vigorous contraction of the recti muscles, produced by the patient rising from the recumbent to the sitting posture without the aid of hands or arms, makes the viscera bulge out prominently in each flank, between the recti and the iliac spines, where the abdominal wall is formed chiefly by the internal oblique and transversalis. When there is a divarication of the recti,

the viscera, instead of bulging out the flanks, protrude in the expanded linea alba. (*n*) The umbilicus is drawn inwards and downwards by the traction of the round ligament of the prolapsed liver. (*o*) In complete enteroptosis the diaphragm is depressed 30 to 50 mm. ($1\frac{1}{4}$ to 2 inches) below the sterno-ensiform line. (*p*) In complete cases the heart is drawn downwards to the extent of an intercostal space—cardioptosis. Corresponding to each beat of the heart and to each inspiration there is a visible tug on the structures at the root of the neck, and the veins fill, instead of emptying during inspiration (Wenckebach). (*q*) The respiratory mechanism is altered. In a late stage, respiration is carried on by the upper part of the thorax, the epigastrium and lower ribs remaining stationary. The action of the diaphragm is irregular in early cases. (*r*) The subcostal angle may be diminished or enlarged. (*s*) The lower part of the thorax is long, funnel-shaped, and narrow from side to side. (*t*) The skin is cold, clammy, and of a greyish hue; the hands and feet readily become cold; the skin of the face is wrinkled and the expression careworn.

Treatment.—1. *Mechanical Support.* The most effective support is obtained by the application of a strapping of rubber adhesive plaster—a method introduced independently by Rose and by Rosewater in America, and by Schmitz in Germany. The rubber plaster must have a thin pliable backing, and be free from resinous substances which irritate the skin. Kemp recommends a rubber adhesive plaster, containing zinc oxide, known in the trade as "30" plaster, and sold in rolls 5 yards long by 7 inches wide. By means of four wide strips cut from the roll, the infra-umbilical wall is strapped to, and supported from, the back, both above and below the crests of the ilium. Before application the abdominal wall must be well cleaned, freed from all grease, and it may be necessary to shave the hair off some of the area to be covered. The length of each of the four plaster straps should be equal to three-fourths of the circumference of the body, measured below the crests of the ilium; each strap being cut so that its anterior or abdominal end has a width of 6 or 7 inches, while its posterior or dorsal end should be rather less than half the width of the anterior end. The straps are applied tightly to the abdomen of the recumbent patient, the broad ends being applied first to the hypogastric region, the narrow ends last, across the back. The two lower or iliac straps, which pass round the body below the crests of the ilium, are applied first. The base of the right lower strap is affixed in front to the left side of the hypogastrium, then carried round below the iliac crest to the back; the base of the left lower strap is applied to the right side of the hypogastrium. The bases of the two straps cross and overlap in front, the narrower ends cross and overlap behind. The two upper lumbar straps are then put tightly on; the base of the right one is applied to the left side of the hypochondrium, carried upwards and backwards just above the iliac crest, pressing into the space between the crest and costal margin, and to support the kidney and liver, and ending by being carried across the

back. The left upper strap is applied in a corresponding manner. Rose uses a fifth strap which encircles the body at the level of the umbilicus; in front the lower border of this strap is cut low so as to descend on the hypogastrium. By a deft use of scissors these straps can be modified as necessity requires. Rose removes the old and applies a fresh support after two weeks; Kemp after four weeks. Patients appreciate these adhesive supports which act by night as well as by day, and cannot be forgotten or misapplied as may occur with abdominal belts. Weissmann has devised a rubber adhesive belt ready for application.

Numerous abdominal belts are available, one recommended by Glénard being much used on the Continent. To be effective they must press the viscera upwards, support the hypogastrium, and at the same time apply pressure to the loin in the region of the kidney. The wearing of such abdominal supports does not induce atrophy or disuse of the abdominal musculature. After having worn one for a month, the patient often finds that he can do without it; it has answered its purpose by resting the musculature of the belly wall and restoring the abdominal circulation. Corsets, if worn, need not be discarded, but they must be supervised, and must be of the "straight-fronted" make, so as to support the hypogastrium. Constriction of the waist must be avoided, as this would neutralise all the benefit of a hypogastric support.

Personal Hygiene.—Rest is to be recommended to the overworked, and activity to the idle and luxurious. Abdominal massage in its various forms assists in restoring the venous and lymphatic circulations. Respiratory exercises and gymnastic movements, planned so as to strengthen and give tone to the muscles of the abdominal walls, may be of service. The exercises should be carried out in the supine position for 20 minutes morning and night. For the purpose of self-massage, the patient may use a six-pound cannon-ball wrapped in flannel, and heated to a point a few degrees above body temperature.

Treatment of the abdominal wall by faradisation, and the use of electrical and other baths, have also been recommended.

Diet.—Einhorn prescribes a liberal diet, with the object of raising the intra-abdominal pressure by the deposit of a layer of subperitoneal fat, the subjects of enteroptosis being always emaciated and malnourished. The condition of the stomach, liver, and intestines requires treatment. Glénard uses laxatives freely; for this purpose natural mineral waters are of service. It must be remembered that the stomach and, in late stages, the intestines are in a state of atony and require bracing by tonics, such as strychnine; this increases peristalsis, which assists the torpid portal circulation. The allowance of alcohol must be strictly limited.

Surgical Treatment.—Operations of many kinds have been tried in incurable cases. Sir F. Treves has reported cases of complete cure effected by operations mainly of an exploratory nature. In enteroptosis, Beyea sutures the stomach to the diaphragm and liver. Duret lifts up and attaches the lesser curvature of the stomach to the anterior abdominal

wall. Coffey elevates the stomach by fixing the great omentum to the antèrior wall of the abdomen. Mr. Eve found the elongated gastro-hepatic omentum so thin that it was impossible to lift the stomach up by reduplicating it with sutures, and even in health, the middle three-fourths of this omentum is usually so weak that it cannot be used for surgical purposes. Gastro-jejunostomy has also been proposed. Surgical treatment of enteroptosis is still in an experimental stage, and should only be attempted as a last resort. When the liver and stomach are lifted up and maintained by sutures, the support obtained is probably not from the sutures, but from the inflammatory adhesions set up by the sutures.

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DISEASES OF THE PERITONEUM

SHOCK

ACUTE PERITONITIS

CHRONIC PERITONITIS

TUBERCULOSIS OF THE PERITONEUM

NEW GROWTHS OF THE PERITONEUM

SUBPHRENIC AND OTHER FORMS OF PERITONEAL ABSCESS

SHOCK

By T. G Brodie, M.D., F.R.S.

Definition.—Shock may be defined as that state of prostration which may follow any excessive disturbance of the nervous mechanism, as in those who have been severely injured, or whose minds have been shaken by intense emotion. A condition closely resembling the more severe cases of shock is known as collapse.

The Causes of Shock.—The usual cause of shock is some severe bodily injury, but strong emotions alone may suffice to bring it about. It may follow violent excitation of cranial or spinal afferent nerves: for example, a disagreeable smell or sight, and is more severe the more sensitive the part injured, as, for instance, the shock following a crush of the fingers, testicles, or larynx. It varies much in severity according to the mental state of the individual, and is markedly influenced by the emotions. Thus, it is well known that soldiers may continue fighting after severe injuries, the excitement of the moment overpowering a stimulation which under other conditions would be quite sufficient to produce shock. Once, however, the mental excitement has subsided, shock appears, and is prone to be all the more severe the longer its onset has been delayed. On the other hand, when aided by excessive emotion, shock may follow quite slight injuries. In this connexion Sir William MacCormac states that the wounded soldiers of a defeated army are more liable to suffer from shock than the equally severely wounded men of the victorious army. In all probability pain is an important factor in the production of shock, but that injury, quite apart from pain or the emotions, may give rise to it, is proved by the fact that anaesthetics have not abolished shock after surgical operations. The great part played by the nervous state of the individual in the production of shock is clearly seen when we study its occurrence in the lower animals. The more highly organised they are the greater is the effect of any specific injury, and the longer the duration of the symptoms. Since nervous excitation plays such an important part in the production of shock, it is easy to understand that the greater the extent of the injured surface the greater is its degree. This is perhaps most obvious in the shock following severe burns, though here another factor, namely, absorption of chemical substances from the damaged cells of the injured surface, may also come into play.

Pathology.—A study of the general phenomena observed in cases of shock indicates quite clearly that the main effects from which the patient is suffering is a notably depressed state of the medulla and spinal cord. That the nervous factor is essentially the chief one is seen from the nature of the causes usually producing shock, and from the marked influence the general mental state of the patient may exert upon the severity and general course of the symptoms. In the consideration of the symptoms it will be seen that some of the most prominent are those in connexion with the vascular system, and consequently they have often been regarded as primary. Thus, one hypothesis has explained shock as primarily due to an extensive dilatation of the vessels of the splanchnic area, brought about reflexly by an action upon the vasomotor centre. If this were so, shock would be, in the first instance, essentially a haemorrhage into the portal area, the blood being held in the large abdominal vessels. But this view is negatived when we notice how greatly the symptoms differ from those of haemorrhage. The part played by abnormal and severe afferent impulses in producing shock has always been fully recognised. The severity of the symptoms is generally proportional to the extent of the injured surface, or to the greater relative richness of nerve-supply to the injured organ. It should be borne in mind that the afferent nerves abnormally excited may be those bearing all forms of afferent impulses, and not necessarily those chiefly associated with one of them, *e.g.* pain. The reception in the cord and medulla of unusually violent afferent impulses leads to a corresponding derangement of function of these nerve-centres, a derangement extending to the medulla and higher parts of the central nervous system. We may regard this derangement of function as primarily due to the reception of an inchoate and excessive series of impulses leading to a simultaneous and irregular excitation of spinal centres, which are not usually thrown into action at the same moment. This view best explains the prominent part which may be played by the state of the cerebrum in initiating or influencing the course of the depression. The more unusual the impulses received the more severe should be the shock produced, as in fact is always the case. Thus, some of the cases of most profound shock follow crushing injuries of the cord.

In considering the mode of production of the more prominent symptoms we should expect to find evidence of the depressed state of the nerve-centres in several directions. For instance, if the spinal grey matter be assailed by an unusual number of impulses, we should expect to find that further impulses of a normal character might go unperceived, and in fact one of the most invariable signs is the difficulty in eliciting a normal reflex. As the next instance the vascular symptoms again receive their best explanation by being attributed to the partial arrest of function of the spinal and medullary vascular centres. The most prominent symptoms are, a feeble circulation, a low blood-pressure, weak pulse, cyanosis, and an accumulation of the blood in the large veins; all of which are best explained as caused by a loss

of nervous control over the blood-vessels and their consequent general dilatation. The physiological study of the vascular mechanism has yielded abundant evidence of the ease with which vascular changes can be produced as a result of afferent nerve stimulation. Thus, electrical stimulation of the central end of the divided sciatic nerve usually produces a rise in blood-pressure due to the excitation of the medullary vasomotor centre and a consequent general vaso-constriction (pressor effect). The continuance of this excitation or an increase in its intensity may soon result in a reversal of this pressor effect and a fall in blood-pressure. Moreover, at times and under conditions which as yet are not fully understood, a stimulation which usually produces a rise in blood-pressure may effect a depression of the vasomotor centre and a fall in blood-pressure (depressor effect). As illustrating this, it has been found that whereas stimulation of the central end of the sciatic in a curarised rabbit causes the usual pressor effect, the result is a depressor one if the animal be chloralised. An extensive series of experiments on the reaction of the vascular mechanism to excessive stimulation of afferent nerves has been conducted by Crile, in an investigation upon surgical shock. He has followed the variations in blood-pressure consequent upon severe injuries of different parts of the body, and has shewn how the severity of the effect depends upon the degree of afferent innervation of the organ injured. If the excitations employed to produce the effects upon blood-pressure be not excessive they are as a rule transitory, and may disappear even though the stimulation be continued. For the production of the effects witnessed in shock it is essential that a more depressed state of the vasomotor centre should be induced, and this is reached if the afferent impulses become excessive and "inco-ordinate" in character.

Examination of the heart during shock does not shew that it is receiving any abnormal nervous impulses, either inhibitory or accelerator, as indeed should be the case if the cardio-inhibitory centre is affected in a similar sense to its neighbour, the vasomotor centre. Weakness of the pulse may be explained simply from the heart not receiving a normal volume of blood. The heart may be perfectly regular in both force and rate. It must of course be influenced by the defective circulation, and as this is much slower than usual, the oxygen supply is deficient, and irregularity and weakness in beat will result in proportion to that deficiency. Further evidence of the defective state of the blood flow may be obtained in various directions. Thus, if an animal suffering from shock be held head upwards, the heart fails to fill, and complete cerebral anaemia results. Also, many of the cerebral symptoms are clearly due to the defective cerebral circulation. The patient is drowsy and difficult to rouse, but not unconscious. The effect of the diminished circulation in the medulla and cord must result in an accentuation of the depression from which they are suffering, and thus we gain one of the main indications of treatment, viz. the necessity of adopting some means of hastening the flow of blood to these parts, that

they may gain an efficient supply of oxygen, the first necessity for recovery.

That the respiratory centre is also profoundly affected is proved by the acceleration and irregularity of the respiration. These effects may be easily produced by excitation of any sensory surface, and are in proportion to that excitation. The feebleness of the respiration will be further accentuated by the defective circulation through the medulla. In addition to these effects upon the respiratory mechanism, an attempt has been made to trace the main cause of shock to a marked diminution or arrest of the gaseous exchanges in the tissues. Thus, John Hunter observed that the venous blood was of a bright red colour in a person suffering from syncope, and more recently Roger has observed the same in cases of shock experimentally produced in animals. This, if confirmed, would be in spite of the markedly diminished flow of blood through the tissues, and would indicate some very serious alteration in tissue metabolism—a change which it would be difficult to ascribe to the general nervous depression. Such a change would lead to a definite decrease of heat production in the body and a consequent fall in body temperature. But though this latter condition is always found in shock, it may be quite readily explained by the increased loss of heat due to the dilatation of the blood-vessels of the skin, and the diminished heat production due to the decreased oxidation in the tissues resulting from the diminution in their oxygen supply. As evidence of the decrease in the activities of the tissues, Roger states that in frogs, in which shock has been induced by crushing the brain, injection of strychnine does not produce convulsions; and it has long been known that, in cases of shock, the administration of opium and alcohol often fails to produce normal effects, though the symptoms may develop later as the shock passes off. These effects have, however, usually been ascribed to defective absorption from the alimentary canal, and Roger's experiments with strychnine have been contested by Contejean. Hence we require further evidence upon the actual condition of tissue metabolism before we can decide how much influence it may be exerting in the production of the phenomena of shock.

Among other conditions which have often been considered as of great importance in the pathology of shock are the changes in the total volume of the circulating blood and the relative proportion of the corpuscles to the plasma. These changes have been chiefly followed by observations upon the density of the blood. In considering these observations it will be advisable to discuss at the same time their bearing upon the allied state of collapse. Collapse occurs mainly in those cases of disease in which loss of water is a predominant factor, and thus while many of the symptoms are practically identical with those observed in cases of shock the effects producing those symptoms may be essentially different. For example, the collapse occurring in the later course of Asiatic cholera is clearly due to the serious loss of fluid occasioned by the severe diarrhoea. The blood becomes inspissated, even so viscid that it has been said to

resemble tar. The features are shrunken and the organs tough, leathery, and of high specific gravity. These changes clearly indicate that the tissues have been drained of their fluid to take the place of that lost by the blood. Such blood changes are not peculiar to cases of cholera, but have been observed in patients suffering from severe and prolonged diarrhoea and vomiting.

Starting from these observations as a basis, a number of experiments have been made in the attempt to determine how far blood changes may occur in cases of shock or collapse. The first observations were those of Prof. Sherrington and Dr. Copeman, who found that the opening of the peritoneal cavity in rabbits was followed by a definite rise in the specific gravity of the blood. A fuller series of experiments was conducted by Roy and Cobbett upon dogs. In these the animals were kept anaesthetised for long periods, and while under the influence of the anaesthetic collapse was brought about by exposing the intestines and handling or ligaturing them. A continuous record of the blood-pressure shewed that for a considerable period the circulation remained unaffected, but that at last it began definitely to fall, and death usually occurred a few hours later. At first the specific gravity of the blood remained unchanged, but in a second period it gradually increased and remained high until the death of the animal. A study of the distribution of the fluids of the body was made by taking simultaneous observations of the density of a normal tissue (skeletal muscle was chosen) and of the injured. From the first, the density of the latter began to fall whilst that of the former rose. Thus, they describe a first period, in which fluid was poured out into the injured tissue while the blood took up water from the healthy tissues, its specific gravity and total volume thereby remaining unchanged, and the general circulation therefore remained normal. In the second period, while fluid was still being discharged into the injured tissue, the supply from the healthy tissues was becoming exhausted, and hence the density of the blood began to rise. This, in its turn, was followed by signs of failure in the circulatory mechanism. In the third period, the effect of this draining of the blood became more pronounced, and finally led to the death of the animal. As was to be expected, the course of these changes was materially modified by the amount of fluid in the tissues of the animal at the beginning of the experiment, available to replace that given by the blood to the injured tissue. They found that in the later stages of the experiments the blood might have lost a very high proportion of its liquid content, even as much as one-third, and consequently only a very diminished volume of blood could be now available for the general circulation. In a series of experiments by Messrs. Ballance and Edmunds, in which portions of the intestine were being resected, the greatest care being taken to damage the intestines as little as possible, Prof. Sherrington again observed a rise in the density of the blood after the operation. A confirmation of the observation was later made by Prof. A. S. Grünbaum in human beings on whom laparotomy had been performed.

There is no question but that loss of fluid from the blood may result in the prostration of collapse, but whether we are to refer all instances of collapse occurring in the course of disease to this initial cause is very doubtful. The similarity of the symptoms observed in shock on the one hand, and in collapse on the other, may depend on a general depression of function of the nervous system.

Symptoms.—These vary according to the severity of the attack. In mild instances there may be a feeling of faintness, pallor of the face, and inhibition quickly followed by acceleration of the heart; then quick recovery. At the onset in more serious cases the patient may stagger and fall, the lips become livid and the skin pale. The respiration may be temporarily suspended, and on returning be of a gasping or sighing character. In extreme cases both of collapse and shock the patient lies in an apparently unconscious condition, with the muscles fully relaxed. If there be any movements, they are irregular, feeble, and jerky. The face is very pallid, lips livid, and the features thin and drawn; the eyes lie sunken in the orbit and the pupils are dilated. Consciousness is rarely completely lost, though much affected. The patient is roused with difficulty, but then will give rational answers to questions. The surface of the body is cold, the skin moist, especially about the head, and the internal temperature subnormal, though in the collapse of fevers the temperature may be raised. To obtain a true value the reading must be taken in the rectum, as the mouth may be cold. The pulse is at first slow, but soon becomes rapid, feeble, and of very low tension. As the general condition gets worse the pulse becomes irregular, and grows progressively weaker until it can no longer be felt at the wrist. The respirations become shallow, irregular, and sighing. The secretions, with the exception of sweat, are diminished or even arrested. The patient suffers severe thirst, the mouth and tongue being dry; the vocal cords are also probably dry, for the voice is feeble, altered in quality, and harsh in tone. The patient feels no pain, the sensibility of the skin is considerably dulled, and the reflexes are difficult to elicit.

Reaction may set in in any time from about two to twenty hours. The pulse improves, especially in volume, and the beat of the heart becomes regular. The temperature rises, and may for a time be above the normal, and the skin becomes warmer. As the depression passes off the patient complains of pain.

Treatment.—If we regard shock as primarily due to an abnormally depressed condition of the medulla and cord, the essential aim of our treatment is to restore the function of these parts to their normal level. First, therefore, the exciting cause, if still present, must be removed, which commonly means the decision whether or no an operation can be safely borne. In the next place, the treatment must aim at alleviating the depression of the nerve centres. Of paramount importance in this direction is the restoration of the circulatory mechanism. As the defect here is chiefly due to loss of tone in the systemic vessels, the position of the patient is of the greatest importance. He should be recumbent,

with the head low and the foot of the bed slightly raised, whereby the return of the blood from the legs and abdomen to the right side of the heart is materially favoured. Bandaging the limbs so as to restrict the amount of blood retained within the veins is often of the greatest service. This principle may also be carried further by applying a fairly tight bandage to the abdomen, care being taken that it is not so tight as to impede respiration. As an extension of this Crile has advocated the use of a pneumatic suit in which the patient can be encased, leaving free the head and thorax. By inflating this rubber suit a pressure may be exerted upon the parts of the body enclosed, thus establishing an artificial peripheral resistance to take the place of the normal one which has been lost. The use of such a mechanism is certainly worthy of an extensive trial. A second valuable line of treatment is to increase the volume of the circulating blood by the administration of saline solution. This may be injected either intravenously or per rectum, the former method being used when a rapid effect is necessary, the latter when an addition to the volume of the circulating fluid is not so urgent. In the former instance the amount injected may be as much as 1 litre of saline solution, made by dissolving 9 grammes of sodium chloride in 1 litre of boiled tap-water. The solution should be injected at a temperature of about 120° F., and must be injected slowly (about ten minutes being taken for the injection of one litre). If necessary, this injection may be followed by a second, and even occasionally by a third. Though this treatment may prove to be of the greatest value, the effect is too frequently transitory. It has been shewn that such injections do not lead to more than a temporary increase of the volume of blood circulating. A subcutaneous injection of saline solution has also been recommended, but can only be of value in cases in which the circulation is sufficiently good to permit a fairly rapid absorption from the injected area. In such cases it is questionable whether a rectal injection is not better.

It is most important that the loss of body heat should be checked, and to this end the patient should be wrapped in hot blankets, and a supply of hot water-bottles should be maintained. Hot baths have also been given with beneficial results, especially in cases of children suffering from burns. Hot coffee and other hot fluids may often be administered with advantage. Doses of ether, brandy, and ammonia are often of service in milder cases. When there is pain an injection of morphia ($\frac{1}{4}$ gr. with sulphate of atropine $\frac{1}{120}$ gr.) may be given, and repeated if necessary. The administration of adrenalin has also been recommended, but I consider that its beneficial effect is only transitory. Strychnine ($\frac{1}{30}$ gr.) has usually been regarded as of great value, but its employment as well as that of stimulants is deprecated by Crile, and with this I entirely agree. In no case must the administration of drugs be pushed, because the delay in absorption, and possibly the defective tissue exchanges, may be masking their actions, which might not attain their maximum until the reaction begins. In cases of collapse from diarrhoea,

water should be given freely by the mouth; or the intravenous injection of saline may be considered. On the whole, collapse should be treated on the same general principles as shock.

T. G. Brodie.

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In the preparation of this article considerable use has been made, in some places, of the article on Shock and Collapse by Dr. Cobbett in Vol. III. p. 320 of the previous edition.

T. G. B

ACUTE PERITONITIS

By W. H. Allchin, M.D., F.R.C.P.

General Pathology and Bacteriology

By F. W. Andrewes, M.D., F.R.C.P.

The Defensive Mechanisms of the Peritoneum.—The peritoneum is by far the largest of the great serous cavities: the area presented by its surface, if it be followed throughout all its folds and recesses, has been estimated as equal to the cutaneous surface of the body. There is little or no free fluid in the healthy peritoneal sac. Its natural moisture is maintained by an exact balance between transudation and absorption. Some degree of transudation doubtless takes place from the whole surface of so vascular a membrane; but attention has been called by Prof. Adami and others to the special secretory functions of the great omentum. There is good evidence that this exceptionally vascular and specialised fold of the peritoneum is truly an "organ" of no small importance in the defence of that cavity, and that a free transudation of fluid from its surface is probably the chief source of peritoneal moisture. It is possible, again, that some degree of absorption may take place through the general peritoneal surface, and in particular through the blood-vessels: there is evidence that this does occur. But there is also conclusive evidence—for instance that brought forward by Muscatello—that the main area concerned in the draining of the peritoneal sac is the concave surface of the diaphragm. It is well known, from the researches of Ludwig and Schweigger-Seidel, v. Recklinghausen, Klein, and others, that the diaphragm possesses a rich and complex system of lymphatics, so disposed amongst the circular and radiating tendon-bundles, that the alternate contraction and relaxation of the muscle in respiration exercises a pumping action whereby a constant current of fluid is drawn up from the peritoneum and passed on into the thoracic duct and retro-sternal lymph-glands. We have thus to conceive of a constant stream of fluid as passing through the peritoneal sac—its main fount rising in the great omentum. Moreover, the pumping powers of the diaphragm are far in excess of what is required in normal conditions, so that they are able to deal with considerable amounts of extra fluid without leaving any residuum behind. In other words, the "absorptive powers" of the peritoneum are known to be very great.

No other serous membrane in the body is so liable to bacterial infection as the peritoneum. It is traversed by 20 or 30 feet of a relatively thin-walled tube, the intestine, the contents of which teem with bacteria to the number of thousands of millions per gramme (Houston). Very many of these organisms are capable of exciting inflammation; they may escape into the peritoneum not only through chance perforations, but by traversing the intestinal wall when this is damaged or inflamed. The gut is thus a standing menace to the sterility of the peritoneum, and there are other hollow viscera which may also rupture into it—the bile passages, the urinary bladder, the uterus and its adnexa. The Fallopian ostia form a potential channel of infection which is ever open. In face of these special dangers it is reasonable to expect that special means of defence against bacterial infections should have been evolved. This indeed is the case. Those general means of defence which are common to all the tissues of the body are fully at the disposal of the peritoneum. The fluid elements of the blood with their load of anti-bodies, the wandering mesoblast with its capacities for phagocytosis—these can be shed into the peritoneum in rich abundance by the aid of the vascular system, and the omentum constitutes an organ by which this can be brought about with exceptional rapidity and thoroughness. These general means of defence against bacteria have been fully described elsewhere [article on the "General Pathology of Infection," Vol. II. Part I. p. 1], and need not be dwelt on further. The peritoneum, however, has certain special advantages. The current of fluid which passes through it is naturally a strong one, and can be greatly accelerated should need arise. Invading microbes can be swept away by this current in purely mechanical fashion, and carried off to the lymphatic glands or blood-stream to be dealt with in a region where their action is less fraught with danger to life. Dr. Durham was able in one case to detect bacteria in the anterior mediastinal lymph-glands six minutes after their injection into the peritoneum. There is perhaps no region of the body where this flushing action is so perfectly developed. Further, the peritoneum is lined by endothelium capable of very rapid proliferation, so that free cells are readily cast off in large numbers into its cavity. These endothelial cells are active phagocytes—less active, perhaps, than the polymorphonuclear leucocytes, but having the great advantage of being on the spot. They constitute one of the chief forms of "macrophage" described by Metchnikoff, and the endothelium of the omentum appears to be peculiarly active in this direction. In the case of those bacteria which can be dealt with by extra-cellular processes of cytolysis (*e.g.* the cholera vibrio), clumping and commencing disintegration of the microbes can be detected within a short time of their injection into the peritoneum of an immune animal, as in the well-known "Pfeiffer's phenomenon." This process, however, seems limited to a few groups of bacteria, and is by no means of general occurrence; nor is it seen except in the specifically immune animal.

These local means of defence seem competent to deal with intruding

microbes, if these are not too numerous or too virulent, without the necessity for evoking the process of inflammation. We may regard them as constituting a *first line of defence*, able to maintain the integrity of the peritoneum against ordinary emergencies. Should they fail, there remains the more potent *second line of defence* in the inflammatory reaction seen in acute peritonitis.

There can be no question as to the general efficacy of the first line of defence, but it is only since the researches of Grawitz that we have come to realise how complete it is. A most exaggerated view formerly prevailed as to the sensitiveness of the peritoneum: no surgeon would interfere with it save under dire necessity. One of the first to raise his voice against this opinion was Wegner, who in 1876 pointed out that this cavity possessed a considerable power of resistance even to putrid fluids, so long as the absorptive powers of the peritoneum were not overstepped. Ten years later Grawitz published the results of a comprehensive and exact research, carried out on careful bacteriological lines. He proved that if care be taken to avoid injury to the peritoneum, non-pathogenetic organisms can be introduced into its cavity in enormous numbers, suspended in water or saline solution, without any injurious effect. He even asserted that pyogenetic organisms, if suspended in a volume of indifferent fluid which did not exceed the absorbing powers of the peritoneum, produced no peritonitis when injected. But if the absorptive powers of the cavity were lessened, inflammation would result: the presence of stagnant fluid, or of wounds of the serosa capable of serving as a nidus for bacterial growth disposed to peritonitis. Many observers repeated these experiments, with various modifications, and on the whole they have tended to confirm the opinions expressed by Grawitz. Mr. Waterhouse, in particular, working under the direction of Orth, obtained almost identical results. It may be taken as well established that saprophytic bacteria, and even facultative parasites of low virulence, if carefully injected into an otherwise normal peritoneum, are got rid of without the production of recognisable inflammation, always provided that the volume of material injected is within the limits of that which can readily be drained away. The first line of defence is adequate in such cases. In one important detail, however, Grawitz's statements have been found to require modification, viz. that which concerns the introduction into the peritoneum of virulent pyogenetic organisms. On this head he was soon contradicted by a number of independent investigators, notably by Pawlowsky and A. Fränkel, each of whom found it easy to produce acute peritonitis by the introduction of quite minute amounts of virulent pyococci into the normal peritoneum. This is now generally accepted, and it is probable that the cultures used by Grawitz were of low virulence. Burginsky shewed this by employing two different strains of *Staphylococcus aureus*: with a virulent strain recently isolated from a case of angina Ludovici he readily produced peritonitis, but with an old laboratory strain no inflammation could be induced in the peritoneum until its virulence had been restored by animal passage.

There is no doubt that the milder chemical irritants are readily diluted and carried off by the peritoneal lymph-stream; the filtered or sterilised intestinal contents can thus be dealt with, leaving no evil effects. With the more intense irritants, capable of causing local necrosis, this is not the case. The recuperative powers of the peritoneum are also manifest against many mechanical injuries, though here the formation of adhesions, or the encapsulation of foreign bodies, may be taken as evidence of a mild and local reactive inflammatory process. But the local reaction, even against the grossest mechanical insults, may be astonishingly small. Von Dembrowski records experiments in which he scrubbed the peritoneum in dogs with a hard toothbrush: no adhesions resulted, and in eight days the endothelium was found again intact, smooth, and shining. Stickler inserted carpet tacks into the peritoneum of guinea-pigs, and subsequently found them encapsuled and rendered practically harmless. Solid material which cannot be digested and broken up seems always to be thus encapsuled. But many solid substances can either be disintegrated and dealt with by phagocytic action, or digested locally by ferments liberated from leucocytes or other cells, and thus such material as blood-clot may be in time removed from the peritoneum.

Enough has been said to make it clear that the first line of defence enables the peritoneum to resist most of the injurious influences to which it is naturally exposed, without the need for anything approaching an acute inflammatory reaction. There is ground for the belief that different regions of the peritoneum vary in their resistance. Thus, the pelvic peritoneum is said to have a specially high degree of resistance. Nevertheless cases are not infrequent, and can readily be imitated by experimental means, in which the ordinary methods of defence are inadequate, and inflammation is evoked as a second line of defence. In this a greatly increased fluid exudate assists the normal flushing action of the lymph-stream, a fibrinous deposit helps to limit the extension of the injurious agency, and a copious emigration of actively phagocytic polynuclear leucocytes supplements the resources of the local macrophages. These processes have been fully described in the article on Inflammation (Vol. I. p. 730), and their details need not be recapitulated here, but the circumstances in which the need for them arises must be taken into account in considering the causation of peritonitis.

The Causation of Peritonitis.—It is certain that the great majority of cases of peritonitis met with in clinical practice are of bacterial origin. There are those who assert that all acute peritonitis is infective; this is open to dispute, but at least it must be conceded that all cases of suppurative peritonitis, the vast majority of cases of acute peritonitis in general, and most chronic forms of the disease, are bacterial in origin. It will be convenient to consider first those forms of peritoneal inflammation which have been regarded as non-bacterial in origin: but it must be pointed out how difficult it is to form an opinion on this point. In a peritonitis primarily bacterial, the microbes may perish, and on culture

the exudate may appear sterile: in a peritonitis primarily non-bacterial secondary infection from the intestine may occur.

(1) The presence of *foreign bodies* in the peritoneum, even though these are perfectly sterile, excites a reaction which would by many be regarded as of an inflammatory nature. As a rule the clinical signs of acute inflammation are absent, and the process is more truly of the nature of repair, belonging to the first rather than to the second line of defence. As examples of this may be quoted cases in which echinococcus cysts rupture into the peritoneum, or in which multilocular ovarian cysts discharge their contents into the cavity. Here the phenomena may in some cases be truly inflammatory, but in others a mere quiescent encapsulation of the foreign material takes place. Meyer records a case in which numerous tubercle-like nodules were found, consisting of cholesterin crystals encapsuled by giant-cells and fibroblasts: the condition originated in the rupture of an ovarian cystoma. In other cases similar nodules have been found to contain hydatid hooklets.

The formation of peritoneal adhesions may similarly be looked upon as a mild localised inflammatory reaction, resulting from mechanical or other injury, and frequently quite independent of bacterial agency.

(2) Cases are not infrequent in which *acute torsion* of an abdominal tumour occurs, *e.g.* of an ovarian cyst or of a wandering spleen. In such cases a peritonitis may occur, and Mr. Malcolm has strongly argued for the aseptic character of such inflammations. In some cases the exudate has indeed been found sterile, but in most of these instances no bacteriological examination has been made. Since the investigations of Dr. Durham and others prove that even with a sterile exudate bacteria may be demonstrable in the omentum, no bacteriological examination can be regarded as complete if this point has been neglected. The observations of Messrs. Dudgeon and Sargent shew that in intraperitoneal haemorrhage a white staphylococcus of low virulence appears in a few hours in the effused blood, and it may well be that this organism is responsible for the relatively mild degree of peritonitis seen in many such cases.

(3) Purely *chemical stimuli* can certainly set up peritonitis. The injection of such substances as croton oil or silver nitrate into the peritoneum in animals is sufficient to produce an acute and fatal sero-haemorrhagic inflammation, but we know of no clinical parallel to this, and many deny a peritonitis in human beings of purely chemical origin. The best instance in man which can be cited is where, from rupture of the liver or bile passages, bile is poured out into the peritoneum in quantity. In such cases a peritonitis has been at times excited which appears aseptic in nature: it is not purulent, but of a benign serous or adhesive character. Boström proved by animal experiment that bile introduced into the peritoneum is relatively harmless. What has been said above as to the determination of bacteria to the omentum, leaving the peritoneal fluid sterile, throws considerable doubt upon the cases regarded as "chemical peritonitis," *e.g.* by Tavel and Lanz.

(4) So-called *idiopathic* peritonitis was at one time regarded as a

distinct clinical entity. A chronic and benign form of inflammation, of sero-fibrinous type, undoubtedly occurs without recognisable cause, in certain rare cases, especially in the female sex and in children. Post-mortem observations are few and bacteriological observations practically absent in such cases. Some are probably tuberculous, though in others no evidence of tubercle can be found. *Rheumatic* peritonitis—another rare condition—may be included here, but in the absence of any conclusive proof as to the causation of rheumatic fever and of any bacteriological evidence in such cases, there is little to be said under this heading. There can be no doubt that many cases formerly classed as "idiopathic" are of bacterial origin, and, when purulent, certainly so. Such obscure suppurative cases are now often spoken of as "*cryptogenetic*." There is no proof that exposure to cold can cause peritonitis, save in so far as chill may act as a disposing cause. The experiments of Grawitz and of Miltner upon chilled rabbits were wholly negative in their result.

Bacterial Causes of Peritonitis.—Many writers endeavour to distinguish between single and multiple infections; but it is almost impossible strictly to classify peritoneal infections on this basis on account of the readiness with which secondary infection takes place. Nevertheless many cases are primarily single infections, and many others multiple from the first, *e.g.* in perforation. Some distinguish a "septic" from a "putrid" peritonitis—the former being due to infection by pyogenetic organisms, the latter by the mixed organisms of the gut. The distinction is not one which can be absolutely maintained.

The following table, taken from Flexner's work, will give a good general idea of the chief bacteria found in acute peritonitis, and of their relative frequency. It is based on the bacteriological examination of 106 cases by that observer: he found bacteria in all but two cases, and was of opinion that even in these two they had probably been present but had died out. Flexner divides his cases into two groups: his *primary* cases are those originating apart from disease of any neighbouring organ, or, in other words, cases due to metastasis by way of the blood or lymph-stream; his *secondary* cases are those arising from injury or disease of an organ in direct connexion with the peritoneum. Further, he divides the secondary cases into *exogenous*, in which the infecting agent is introduced from without the body, and *endogenous*, in which it comes from within.

[TABLE

Variety of Bacterium.	Total Cases in which found.	Alone.	In Combination.
Exogenous cases—			
Staphylococcus aureus	15	12	3
Staphylococcus albus	3	2	1
Streptococcus pyogenes	10	5	5
B. coli communis	7	2	5
Pneumococcus	3	1	2
B. proteus	1	0	1
B. pyocyaneus	2	0	2
Undetermined	3	0	3
Endogenous cases—			
B. coli communis	47	9	38
Streptococcus pyogenes	39	7	32
Staphylococcus albus	4	2	2
Staphylococcus aureus	3	1	2
Pneumococcus	4	1	3
B. proteus	4	2	2
B. aerogenes capsulatus	8	2	6
B. pyocyaneus	3	0	3
B. typhosus	3	0	3
Undetermined	3	0	3

The results obtained by other observers are in practical accord with those of Flexner. It will be noted that organisms known to be saprophytic in the normal alimentary canal play a part of overwhelming importance in the "endogenous" cases, and are prominent even in the "exogenous" series. *Bacillus coli* heads the list, and *Streptococcus pyogenes* comes next. Flexner seems to have regarded all streptococci as this organism, but it is certain that he must have included here a great majority of intestinal streptococci which have no right to the name *Streptococcus pyogenes*. With the exception of *B. typhosus* (and possibly of *Staphylococcus aureus*, concerning which no data are forthcoming) practically every microbe in Flexner's list is known as a faecal saprophyte.

The various bacteria commonly occurring in peritonitis must now be considered in further detail.

Staphylococci.—*Staphylococcus pyogenes aureus* has been found by all observers as a not infrequent cause of peritonitis, and the inflammation which it excites is of a very serious and fatal character. Death may result before the peritonitis has had time to assume the suppurative form which this organism usually induces. *Staphylococcus albus* may at times be but the white variety of the preceding organism, but the work of Messrs. Dudgeon and Sargent has shewn that the common white staphylococcus of peritoneal inflammation is a perfectly distinct organism, and there is good reason for believing it to be identical with the common microbe of the skin described by Welch as "*Staphylococcus epidermidis albus.*" It differs from the true *Staphylococcus pyogenes* in its feeble pathogenicity, and the relative slowness with which it liquefies gelatin, and above all, as Dr. M. H. Gordon has shewn, by its inability to ferment

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mannite. Messrs. Dudgeon, Sargent, and Ross have proved that it may be found in the omentum of normal guinea-pigs, that in intraperitoneal haemorrhage in human beings it speedily appears in the effused blood (only in one case out of twenty was it absent), and that it is common in the majority of cases of peritonitis due to the most various causes. They go farther than this in suggesting that this organism, by its early appearance in peritoneal inflammations, may actually play a conservative part—"preparing" the peritoneum, as it were, by stimulating the defensive agencies, for the advent of more serious subsequent infections. In experiments upon guinea-pigs they were able to shew that preliminary intraperitoneal injections of this coccus, twenty hours before injection of *B. coli*, had a truly beneficial effect, either saving the animal's life or much delaying its death.

Streptococci.—There can be no doubt that these are amongst the commonest and most important pathogenetic agents in peritonitis, almost ranking with *B. coli*; and it is noteworthy that these two groups of organisms are the most abundant saprophytes in the alimentary canal. It is difficult to make any precise statements as to the species of streptococcus found, because it is only quite recently that any tests have been discovered by which the different forms can be separated. Nearly all observers have classed any streptococcus found as *Streptococcus pyogenes*, and here they have certainly been in error. The true *Streptococcus pyogenes* does indeed occur in peritonitis, and gives rise to the most dangerous and uniformly fatal form of the disease: I have met with it several times in post-operative cases. But more commonly, and especially in perforative peritonitis, the streptococci found are of feeble virulence, and appear to be identical with the short-chained forms common in the alimentary canal. I have repeatedly found them absolutely identical with these in their chemical reactions. The true *Streptococcus pyogenes* has not hitherto been recognised in normal faeces. Dr. Houston tested a very large number of faecal streptococci upon mice and found no single one pathogenetic. When, in perforation of the intestine, the mixed faecal organisms gain access to the peritoneum, it is not uncommon for these feebly virulent streptococci to die out in a short time, while the more hardy and vigorous *B. coli* flourishes, and may ultimately be found, as Barbacci pointed out, in pure culture. They do not always perish in this fashion. I have isolated a typical faecal streptococcus, in company with *B. coli*, from an empyema secondary to a perforated appendix. It is probable that even when they do perish they have already played no unimportant part in the causation of the inflammation induced. It is not unlikely that the mixture of organisms may be more effective than any one alone. It would seem that in perforations of the stomach and duodenum, in which *B. coli* plays an insignificant part, the peritonitis is often more benign and localised than in perforations lower down in the intestine. In such cases the saprophytic streptococci of salivary type may be present almost alone, as I have more than once found. In gastric perforation Messrs. Dudgeon and Sargent describe as almost constant a "strepto-diplococcus"

which is presumably a short-chained streptococcus, though negative to Gram's stain.

The Pneumococcus has now for many years been recognised as an important though not very common cause of peritonitis. It has been found in cases of gastric perforation and in appendicitis, and also in not a few cases of spontaneous suppurative peritonitis, formerly regarded as idiopathic. Pneumococcal infection rarely spreads from the thorax to the peritoneum, and its frequency below the diaphragm is small in comparison with that seen above that partition. There are two reasons for this: the pneumococcus is a much more characteristic saprophyte of the respiratory than of the intestinal tract; and the direction of the lymph-stream is from the abdomen to the thorax, so that while pleurisy is a common complication of peritonitis, the reverse infection is comparatively rare. Pneumococcal peritonitis is usually suppurative, acute and severe in onset, but amenable to early surgical treatment. Being fibrinous in character it often becomes localised.

The Gonococcus.—Gonorrhoea is responsible for a considerable number of cases of peritonitis, especially in the female sex, but comparatively few of these are actually due to the gonococcus alone. As a rule the peritonitis is due to secondary infection by some other organism; nevertheless, a number of cases have been proved bacteriologically to be truly gonococcal in nature, and such cases seem usually comparatively benign.

Bacillus coli is the commonest microbe in peritonitis, and this for several reasons. As one of the most abundant saprophytes of the intestine, it is universally found in peritonitis from intestinal perforation; having got into the peritoneum its vigour and hardiness enable it to maintain its footing there better than many other organisms which may also have been present at the first. It can also gain access to the peritoneum through the damaged intestinal wall in acute circulatory disturbances, *e.g.* strangulated hernia, and even through the merely inflamed gut where the primary mischief is due to other bacteria. Thus, it is a common secondary, as well as primary infection. This is well exemplified by some experiments carried out by Dr. E. Klein. He inoculated guinea-pigs intraperitoneally with *B. prodigiosus*, which can cause acute peritonitis in these animals; he used the peritoneal exudation of the first animal for injection into a second, and carried on the process through a series of guinea-pigs. Plate-cultures were made from the exudate in each case. *B. coli* appeared in increasing numbers in the series, and finally entirely replaced the *B. prodigiosus*. The injection of pure *B. coli* into the peritoneum in rodents suffices to set up a rapidly fatal peritonitis, and this, as Dr. Klein has shewn, even if the culture injected has previously been sterilised by heat, though here larger doses are required. This must mean that the bacterium contains an intracellular toxin. In many human cases of peritonitis *B. coli* is found in pure culture, but this must not be taken to prove that no other organism was originally present. Barbacci has strongly insisted on this, and has recorded an instructive experiment bearing on the point. He produced intestinal perforation in five dogs by

means of a caustic, closed the abdomen, and then examined the animals after 5, 5½, 6, 8, and 10 hours. In the four animals earliest killed he found a mixture of organisms present in the peritoneum, but in the animal killed after 10 hours *B. coli* alone was present. The survival of this organism in such cases must depend upon its more pronounced capacity for facultative parasitism—in other words, its greater power of resisting the defensive mechanisms of the peritoneum. A. Fränkel was able to produce in animals various forms of peritonitis by the injection of pure cultures of *B. coli*. At times they produced a mere transitory constitutional disturbance, or again an acute general infection with speedy death but little local reaction; in other cases, however, there was a pronounced local reaction—either a diffuse fibrino-purulent peritonitis or multiple sacculated abscesses in the peritoneum. This corresponds fairly with what has been observed in human peritoneal infections by this organism which are by no means uniform in type. Much, doubtless, depends upon the virulence of the invading organism, but on the whole *B. coli* does not set up in man such intensely severe peritonitis as is seen in infection by *Streptococcus pyogenes*. Mixed, however, with other organisms, and with the contents of the intestine, the inflammation may be very virulent. It is customary to regard the form of the colon bacillus found in peritonitis as *B. coli communis*, and in most cases this is probably correct, since the classical *B. coli communis* is the prevailing intestinal type. Nevertheless, other members of the colon group are common in the intestine, and it must not be assumed that any given organism in peritonitis is the true *B. coli communis* unless it correspond in all its chemical reactions.

Many other organisms have been met with in peritonitis, but the ones above described are indisputably the most important. In peritonitis spreading from the intestine, by perforation or in other ways, any intestinal organism is liable to be found. Thus, *B. pyocyaneus*, *B. proteus*, and various anaerobic bacilli have been found, such as *B. aerogenes capsulatus*. The part played by anaerobic bacilli in the production of peritonitis is very imperfectly known, because anaerobic cultures have so rarely been employed in investigations on the subject. It is not improbable that they are of more importance than has commonly been assumed. Friedrich has insisted on this. He found them almost constantly present in peritonitis from perforation and gangrene of the gut, and of several species. The typhoid bacillus has been found in the peritoneum in typhoid perforation, but there is nothing to shew that it plays any part more important than that of the much more abundant intestinal saprophytes which escape with it.

Disposing Causes of Peritonitis.—In what has been stated above as to the bacteria found in peritoneal inflammations, no mention has been made of the conditions which aid the bacteria in their attack. Given a sufficiently high degree of virulence in the bacterium, no disposing cause is needful. The more virulent forms of pyogenetic cocci, and notably *Streptococcus pyogenes*, require no adventitious aid. Experiment

indeed proves that mere residence in the peritoneal cavity greatly increases the virulence of these organisms, and the method of successive passages through this cavity in a series of animals has been largely used by bacteriologists to increase the pathogenetic powers of various microbes. Many of the graver instances of infection at post-mortem examinations have been from cases of septic peritonitis due to *Streptococcus pyogenes*.

But in the case of organisms of lesser virulence, such as the merely facultative parasites from the alimentary canal, disposing conditions play a part of no small importance, as has been proved by the observations of many different experimenters upon the animal body. The animal experiments are supported by a number of clinical facts as regards human peritonitis. Any condition which hinders absorption from the peritoneal cavity gravely impairs its first line of defence, leading to the presence of a stagnant fluid in which bacteria can multiply. Any preliminary injury to or disease of the peritoneum may act in this way: ascites is thus a disposing cause, and peritonitis is apt to occur in the subjects of chronic nephritis. In the next place, the presence in the peritoneal cavity of foreign material, not readily absorbed, may provide bacteria with a breeding ground. This is seen when blood has been effused there. Mention has already been made of the observations of Messrs. Dudgeon and Sargent as to the speedy appearance of a white staphylococcus, even when the effused blood may be assumed to have been originally sterile. Much more must the clot favour bacterial growth when organisms are abundantly present from the first. The contents of the alimentary canal, escaping into the peritoneum, have undoubtedly a similar effect, so that their mere mechanical presence plays a prominent part in the production of perforative peritonitis. Barbacci lays stress on the intestinal gases in this connexion, and brings forward some experimental evidence in support of his contention. He also points out the great importance of the continuous reinfection of the peritoneum in perforative cases by the frequent passage of intestinal contents through the aperture into the already inflamed cavity. He experimented on five dogs, closing an artificial perforation in the gut after a varying number of hours, and proving that early closure had a beneficial effect. Lastly must be mentioned traumatic disposing causes other than perforation. Grawitz has strongly maintained that peritoneal wounds of all kinds favour the production of peritonitis by affording a nidus for bacterial growth, and many other subsequent observers have confirmed this, though simple well-apposed wounds may have little effect. The tearing of adhesions during intraperitoneal operations may thus have injurious consequences. Favoured by one or another of the disposing conditions which have been mentioned, it is well established that many facultative parasites which produce no inflammatory reaction in their absence can set up a peritonitis of severe nature.

The Cytology of Peritonitis.—The normal fluid of the peritoneum is extremely scanty, but contains free cells of several distinct varieties. All observers are agreed as to the frequency of occurrence of two kinds of cell, viz. the lymphocyte and the large hyaline cell, which is pre-

sumably of local endothelial origin. Messrs. Dudgeon and Ross estimate this latter cell at 70 per cent of the total, and the lymphocyte at 25 per cent. More doubt exists as to the oxyphil cells. Kanthack and Hardy asserted that finely and coarsely granular oxyphil cells (polymuclear neutrophils and eosinophils) formed 30 to 50 per cent of the total, but later observers have not confirmed this. Dr. Durham admitted the eosinophil (megoxyphil) cell as normal to the peritoneum, but was unable to find the polynuclear neutrophil (microxyphil) under normal conditions. Messrs. Dudgeon and Ross could find neither variety. Kanthack and Hardy describe also small numbers of unstable basophil cells as present. There can be no doubt that the varying results recorded depend in part upon the varying nature of the cells in the different species of animal examined: most of the work has been done on guinea-pigs and rabbits, but on the whole the cells in man seem to correspond fairly with those seen in these animals.

One of the most important contributions to the cytological study of peritoneal infections is that of Dr. Durham, and his results are in substantial accord with those of later observers, *e.g.* Messrs. Dudgeon and Ross. He found that the sequence of events, when a non-fatal dose of bacteria was injected into the peritoneum of animals, was as follows. In from two to five minutes the normal hyaline and eosinophil cells of the peritoneal fluid became clumped into balls: the lymphocytes remained free. Save for these latter cells, the peritoneal fluid quickly became almost free of cells, and this stage of leucopenia lasted for about an hour. He does not agree with Metchnikoff and with Kanthack and Hardy that this phenomenon is due to disintegration of the cells, but regards it as due to the adhesion of the viscous balls of cells to the peritoneal surface, and, in particular, to the great omentum: he gives good reasons for this belief, which has since been confirmed by others. During the leucopenic stage the amount of fluid in the peritoneum shews a marked increase. Phagocytosis is abundantly present even in this leucopenic stage, provided that the organism injected is not one of high virulence: in the case of very virulent organisms it is feeble or absent. This too is in harmony with the observations of others; thus, E. C. Rosenow has found that the polymorphonuclear leucocytes of the blood are unable to ingest virulent pneumococci while fully able to deal with those of feeble virulence. The phagocytes during the leucopenic stage are mainly the hyaline (endothelial) cells, and they are active despite their clumping and adherence to the peritoneal surface. It was even found that cultures could readily be obtained from the omentum (in later stages) when the peritoneal fluid was sterile. About an hour after the injection a new factor appeared upon the scene, namely, the advent of polymorphonuclear leucocytes, derived from the blood-vessels. These increased in number up to 15 hours, the fluid decreasing in amount and becoming turbid. These cells are even more efficient phagocytes than the hyaline cells, and, according to Messrs. Dudgeon and Ross, play an even more important part in freeing the peritoneum from micro-organisms. Dr. Durham found that in

15 or 16 hours the "macrophages" of Metchnikoff began to appear in increasing numbers, derived undoubtedly from endothelial proliferation, and especially from the omentum. These cells, in a non-fatal infection, complete the task, devouring not only bacteria but even the polymorphonuclear leucocytes, of which it is common to find several ingested by a single macrophage. In from 4 to 6 days the peritoneum then gradually returns to the normal.

The part played by the eosinophil cells has been variously assessed by different writers. Kanthack and Hardy maintained that in the frog these cells played a preliminary part, discharging their eosinophil granules upon the bacteria and in some way damaging these so as to prepare them for ingestion by the more actively phagocytic cells. Dr. Durham, and also Messrs. Dudgeon and Ross, are unable to confirm this. They admit an increase in the eosinophils in many cases, but found no good evidence of the discharge of granules, and it seems clear that these cells have occasional, though feeble, phagocytic powers. In experiments extending over more than 80 animals, Messrs. Dudgeon and Ross found that eosinophils formed over 15 per cent of the peritoneal cells in 20 cases, and this in the case of infection with the most widely different bacteria, and even after injections of normal salt solution and emulsions of chalk.

Such are the chief cells which play a part in acute peritonitis. The activity of the endothelium is manifested by the rapid budding of the cells, especially on the omentum. It should be added that in tuberculous peritonitis, as in this form of inflammation in other serous cavities, the small lymphocyte is found as the predominant cell in all but early cases. Mr. E. A. Ross found them as high as 80 per cent in the peritoneal fluid of some guinea-pigs which had been inoculated with fluid from tuberculous pleurisy. The preponderance now of one form of cell, now of another, depends doubtless in part upon the stage of the inflammation, and in part upon the special chemiotactic properties of the invading bacterium. In the case of the ordinary pyogenetic organisms a purulent inflammation ensues if sufficient time elapse, because of their intense chemiotactic influence upon the polynuclear leucocytes. But even here death often ensues so rapidly that there is no time for suppuration in the ordinary sense of the term. In many cases of supposed shock and collapse after laparotomy, cultures from the peritoneum and blood may yield *Streptococcus pyogenes*, though to the naked eye the peritoneal changes appear trivial.

There are other features of acute peritonitis which demand some notice. One is the formation of a fibrinous coagulum from the exuded fluid: this may be of vast service in checking the spread of micro-organisms, for the temporary adhesions thus brought about are speedy and far in advance of permanent organised ones. In appendicitis or in threatened typhoid perforation such fibrinous exudates may preserve life by warding off a general peritonitis should perforation subsequently ensue. There are other forms of peritonitis in which red blood corpuscles pass in numbers into the peritoneal cavity, leading to the haemorrhagic

form of inflammation. In yet others, the bacteria present (especially *B. proteus*, and anaerobic bacilli of intestinal origin) may cause putrid changes in the effused fluid, whence may arise the foul and putrid forms of peritonitis, sometimes with evolution of gas into the peritoneal cavity.

Lastly must be mentioned the formation of organised adhesions, so common a feature in the later stages of peritoneal inflammation. The histology of these in no way differs from that seen in other serous membranes: it is a question of the development and vascularisation of fibrous tissue. The chief matter in dispute concerns the nature of the cells which develop into the fibrous tissue, and here there are two distinct opinions. On the one hand it is maintained that the peritoneal endothelium is capable of a metaplasia into connective tissue, on the other that the fibrous tissue is formed by the immigration of young cells derived from pre-existent cells belonging to the fibrous tissues and not to the endothelium. Ranvier held that an immediate union of serous surfaces could take place by the direct conversion of endothelium into fibrous tissue; many later writers have shared this opinion, wholly or in part, *e.g.* Marchand, Graser, Roloff. On the other hand, Hinsberg concludes that the peritoneal endothelium never has a genetic connexion with connective tissue. Rissmann found that layers of intact endothelium can adhere to one another, even in the absence of bacteria and mechanical irritation, but only loosely: for firm union loss of endothelium was necessary. Even Graser admits that the commonest form of peritoneal adhesion is by union of the sub-endothelial layers after loss of the surface endothelium. The occurrence or non-occurrence of a direct metaplasia of endothelium into fibrous tissue is thus not finally settled, but it is at least probable that the more important elements forming permanent fibrous adhesions are the descendants of connective-tissue cells. In the case of aseptic foreign bodies in the peritoneum, the surrounding cells may shew large syncytial masses or giant-cells: the endothelial origin of such masses is not improbable.

In conclusion, a word may be added as to the pathological basis of the symptoms and upshot of cases of acute peritonitis. After what has been said as to the drainage of the peritoneal cavity into the lymphatic system and thus indirectly into the blood, it is no matter for surprise that a septicaemia should be present as a terminal event in a large number of cases. Streptococci, colon bacilli, and other organisms are recoverable from the heart's blood in many, perhaps in all, cases of fatal acute peritonitis. Libman, however, who examined the blood of some 25 cases of peritonitis during life, was unable to find bacteria in any instance, though in some cases the examination was made shortly before death. In some of the most rapidly fatal cases the peritoneal lesion is trifling to the naked eye, though manifest enough to the bacteriologist: the disease is essentially a septicaemia, with the peritoneum as a portal of entry. Conversely, a peritonitis is a common secondary event in any acute septicaemia. In other cases of peritonitis the constitutional

phenomena appear rather of a toxic nature dependent upon the absorption of decomposition products from the peritoneum. Barbacci has studied the histological changes in various organs in perforative peritonitis, and finds diffuse and focal necrotic changes in the liver, spleen, and lymphatic glands, but especially in the kidney, which he attributes to absorbed toxic products. The fatal event in acute peritonitis may thus depend either on septicaemic or toxic conditions.

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Acute Diffuse Peritonitis[1]

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Channels of Microbic Invasion. — Accepting the cause of acute peritonitis in man to be bacterial infection, the question next arises by what means or by what channels does the invasion by the micro-organisms take place?

[1] By the term "diffuse" is meant a peritonitis which is not limited to one region of the abdomen by adhesions, but spreads more or less over the entire cavity until it may become "general." As this latter condition can only be ascertained by inspection, either at operation or after death, and is but the most completely diffused form, the expressions "diffuse" and "general" will be used without distinction in the following article.

(*a*) The simplest and most obvious way in which the pathogenetic microbe may obtain entrance to the peritoneal cavity is by a stab or similar injury that shall have penetrated the abdominal wall and established a communication with the serous sac. If the viscera should have escaped damage by the wound, the weapon and the passage it has made may be fairly regarded as the means of infection *ab extra*; whilst if, for instance, the stomach or intestines are also injured the likelihood of a peritonitis being set up by invasion from the alimentary tract is proportionately increased, and more especially if the injury have gone beyond a mere contusion of the viscus and have caused perforation. "Some experiments on animals shew that under specially favourable conditions peritonitis does not necessarily follow the traumatic form of intestinal perforation (for instance, when the perforation is closed by the omentum, or when the perforated bowel is empty and becomes vigorously contracted): in clinical practice this accidental prevention of secondary peritonitis is so rare that it can hardly be taken into consideration" (Nothnagel).

(*b*) That acute peritonitis may occur in connexion with various inflammatory conditions of the abdominal viscera but without obvious breaches of continuity of the organs is sufficiently familiar. Without doubt appendicitis is the most frequent cause, when the defensive measures on the part of the peritoneum have failed to confine the results of the appendicular mischief to the immediate neighbourhood, as may be the case when the infection is of peculiar virulence or when a fibrinous exudate has not been formed in response to the irritant. Ordinarily an invasion of the peritoneum by organisms, that make their way through an inflamed appendix, gives rise to a localised peritonitis or abscess, sufficient time in such circumstances being afforded for the requisite protection. The various forms of acute intestinal obstruction are most likely to lead to a peritonitis that may be general, or, if more gradually induced, of a localised character. A bacteriological examination of 100 cases of intestinal obstruction by Messrs. Dudgeon and Sargent shewed the presence of micro-organisms in nearly half the cases. Separating these into two groups of 47 cases of strangulated hernia, and 53 of other forms of occlusion, they found that 33 per cent of the former were infective, micro-organisms being obtained either from the fluid in the sac or from the surface of the bowel, and 56 per cent of the latter yielded some form of pathogenetic microbe. But, whilst in strangulated hernia there is a steady increase in the proportion of infected cases with the increase in duration of the strangulation, among the other forms the proportion was markedly higher in the acuter cases. They explain this discrepancy by supposing "that the more frequent sterility of strangulated hernia is due to the relatively smaller area of intestine available for examination," for they believe that the organisms escape not only at the site of most severe damage, but also from all parts of the bowel as it becomes inflamed and distended. Further, they attribute the more acute symptoms observed in obstruction of the small as compared to the large intestine as being due in part to the fact that peritoneal

infection takes place more rapidly from the former than from the latter. In the less acute cases they regard the peritoneum as possessing the power of dealing with the organisms that appeared in the earlier stages, and hence such cases are more frequently sterile. By far the most frequently occurring organisms were a white staphylococcus and the *Bacillus coli*.

A number of cases (10, 21) have been recorded of a general peritonitis supervening upon enteric fever without any perforation, or coincident disease of the appendix, the pathogenetic organisms having traversed the walls of the damaged intestine. It also appears that all but the mildest degrees of inflammation of the gastro-intestinal mucosa are capable of so diminishing the resisting power of the walls of the canal as to permit the passage of the infective agents, and it is possible that the same result may follow from severe cholecystitis, although it is more usual that a chronic form of peritonitis with the local formation of considerable adhesions should then be established.

Among the exceptional causes of peritoneal inflammation to be included within this group are suppurative pylephlebitis, and phlebitis of the umbilical vein following on infection soon after birth—the usual cause of peritonitis in the new-born.

(*c*) The most acute and severest cases of diffuse peritonitis are attributable to perforation or rupture of the hollow viscera or the bursting of an abscess, with escape of their contents into the peritoneal sac. Here again the appendix is by far the most common source of the mischief; perforative ulceration or gangrene of this structure being of frequent occurrence. Next in order of frequency are perforation of the stomach, duodenum, and ileum, concerning which it may be said that gastric ulcer is oftenest the cause; the corresponding lesion in the duodenum owing to the anatomical arrangement of the first part of that organ, where the peptic ulcer is usually situated, may, if on the posterior wall, lead to a localised abscess in the post-peritoneal tissue instead of bursting into the general peritoneal cavity. Among the various causes of intestinal ulceration much difference exists as to the liability to perforation, the typhoid lesion being that most prone, although as the result of the operation of gastro-enterostomy a considerable number of perforating peptic ulcers have been recorded in the small intestine. In general it may be stated that the infection of the peritoneum from the intestine is clinically more virulent in character than that from the stomach, the contents of which, when consisting only of the normal secretions, may be almost or quite sterile, and hence it is that recovery may have followed from gastric perforation. Malignant growths in the course of their destructive advance may determine a rent in the wall of the bowel, oftener in the large than in the small intestine, but this accident is commonly gradual in occurrence and thus allows time for localisation of its effects by adhesions. The gall-bladder and main bile-ducts, the pancreatic duct, or the ureter may also give way under conditions of inflammation and ulceration extending outwards from their mucous surfaces.

As the result of obstruction or gangrene the bowel may rupture; this

may also happen to a pyosalpinx or to a suppurating ovarian or other cyst with consequent extensive infection of the peritoneum. A like result will follow from the bursting of an abscess of the liver, pancreas, spleen, or kidney, or of a suppurating lymphatic gland, or of a perimetric abscess, or of an empyema or pulmonary abscess that has extended through the diaphragm.

(*d*) Due to the special anatomical condition in the female permitting communication between the peritoneal sac and the exterior by the open ends of the Fallopian tubes; it is desirable to consider peritonitis caused by infection via this channel separately, since it is responsible for the majority of the cases of peritonitis in females. The liability to inflammation of the genital passages in females, and particularly to that form determined by the gonococcus, menstruation, and parturition, are each and all circumstances that in different degrees favour the occurrence of peritonitis, both acute and chronic, general and local.

It will be observed that in all the foregoing cases the onset of peritonitis is secondary to mischief elsewhere, and the question now arises whether a primary—or as it has been commonly termed idiopathic—inflammation of the serous membrane can occur. Before the bacterial nature of the malady was understood, many cases were regarded in this light, but with increased knowledge the limits of the group have very considerably shrunk until there are those who now deny the existence of any but secondary cases. It must be admitted, however, that, for want possibly of more precise information, cases of peritonitis now and then occur which cannot be referred to any of the groups already indicated. But in those exceptional instances in which even examination after death fails to reveal a source for the infection of the peritoneum, it must be remembered that microscopic damage to the wall of the bowel and especially of the appendix may suffice to allow the passage of pathogenetic organisms, and on the other hand that such organisms after setting up the inflammation may be so disposed of by the peritoneum as to give no evidence of their existence, with the result that the peritonitis is looked upon as idiopathic and of chemical causation; assumptions that cannot be definitely proved or disproved, but that are in the highest degree improbable in view of known facts.

The association of peritonitis with the exanthemata and other acute infective diseases, among which is to be included rheumatic fever—an extremely rare occurrence,—was formerly looked upon as being much closer than present-day experience warrants. In some of these affections the intestines or other viscera are occasionally infected, and the explanation of any peritonitis that may arise is obvious; or it may be that there is some local lesion in the lungs or pleurae whence invasion through the diaphragm may possibly take place (*vide* also p. 899).

Morbid Anatomy.—A condition of disease which is attributable to such diverse causes as is acute peritonitis, which exhibits such differences in its onset, extent, and course, is likely to manifest a considerable range of variation in the structural changes met with: and so it is. Nevertheless,

in their main features the morbid appearances conform to such as obtain in the inflammation of the serous membranes generally. It may be premised that certain discrepancies appear in the accounts given by different authorities, and that these would seem to depend upon the conditions under which the observations were made—whether the appearances recorded were such as were recognised during life when the abdomen was opened, or whether they were described from what was seen after death.

The first and most obvious departure from the normal to be found is a state *of hyperaemia* of the serous membrane, most marked in the neighbourhood of the causal lesion, and to a much greater extent over the mesenteries, the omenta, and the visceral layer of the peritoneum, than over the parietal portion, and especially prominent along the intestinal coils where they are in contact with each other. It is not always easy accurately to gauge the degree of the hyperaemia present in any case, since this gives rise to a totally different appearance before death to what is presented afterwards, and it should be estimated by what is apparent when the abdomen is first opened during life, for the exposure and handling of the viscera quickly determine an increased vascularity quite apart from that due to the inflammatory state. In addition to the injection, it is not unusual for small haemorrhages to be seen in or immediately beneath the serosa. In the fulminant cases of the disease nothing beyond these vascular appearances is to be found, death taking place before there is any visible change in the tissue elements.

The peritoneum in the earliest stages loses its healthy glistening appearance, and assumes a dull, opaque, greyish look. It becomes swollen and oedematous, so that it can be stripped off the subjacent structures with greater facility than in the normal state. The endothelial cells undergo cloudy swelling with proliferation of the nuclei, and, being shed to a greater or less extent, thus give rise to some of the phagocytic elements found in the exudate, leaving the peritoneal surface proportionately denuded. The lymph-spaces are filled with fluid and the subjacent tissues shew a general small-celled and serous infiltration which extends to a variable depth into the substance of the organs covered by the serous membrane. The cells are derived partly from the leucocytes of the blood and partly from the division of the connective-tissue corpuscles; some red blood-cells and fibrin can also be recognised. These changes are most prominent over the omentum, and somewhat less over the visceral layer generally, spreading obviously from the site of entrance of the infection.

As a result, no doubt, of the greater severity of the inflammatory process in the peritoneum covering the intestines, these organs shew, as a rule, more in the way of morbid changes than do the other viscera. The tissues of the intestinal wall, apart from round-celled infiltration which may extend through into the mucosa, are oedematous and swollen, becoming soft and very easily torn. Sometimes the muscular fibres are degenerated and necrotic, but, except for their greater friability, are often not manifestly altered; but degenerative changes in the intrinsic nerve-ganglia and plexuses have been demonstrated. These alterations in the

intestinal wall may be in part responsible for the distension and paralysis of the intestines which are such striking characteristics of the disease.

Of the inflammatory products the earliest to appear is a fibrinous film disposed more or less completely over the inflamed area, ranging in amount from such as may lead to a mere stickiness of the surface to patches of considerable thickness, filling the interstices between contiguous coils of intestine and forming layers over the liver and spleen. The adhesions between the various structures may be of the slightest, or, if several days have elapsed, may be sufficient to require some force to separate the cohering parts. Of a yellowish colour, the surface of this "inflammatory lymph" may be rough and honeycombed, whilst flakes of similar material are free in the fluid exudate. The blood is the chief, if not the sole, source of this material, which leaves the capillaries as a coagulable plasma and then provides the fibrin elements. Neumann and others have considered that another source of the fibrin is the sub-endothelial connective tissue, which is described as swelling up and becoming hyaline, undergoing what is termed a "fibrinoid degeneration." (See art. "Inflammation," Vol. I. p. 766.) It is important to realise that this fibrinous layer has a distinctly protective and beneficial function. The absorption of toxic material from the peritoneal cavity is more or less arrested by the interposition of fibrin between the serous sac on the one hand and the blood-vessels and lymphatics on the other. Further, by the coating it forms, it preserves the endothelium, upon the integrity of which depends the subsequent absorption and removal of the adhesions formed by the "lymph," a point of no small moment.

Together with the fibrinous exudate there is poured out a fluid exudate seldom very large in amount, and usually in quantity from ten to thirty ounces—sometimes even less. In character the fluid is serous, of a specific gravity of about 1015, and containing 2 to 5 per cent of protein. Seldom perfectly clear in appearance, it is generally more or less turbid, from the presence of leucocytes and granular débris up to the condition of being purulent, although pus occurs more commonly in localised peritonitis than in the diffuse form. A spontaneously coagulable fluid, flakes of fibrin are found in it, enmeshing the cells, forming yellowish or yellowish-green masses of all sizes. (For the cytology of peritonitis, *vide* p. 905).

Besides the several appearances already mentioned, the exudation may be more or less deeply blood-stained, especially when the cause of the peritonitis is one associated with much congestion of the serosa, such as intussusception or strangulation of the bowel or, as will be subsequently noticed, in the presence of tuberculosis, or more especially of malignant disease. When the inflammation is consequent on gangrene of the appendix or other viscus, the effusion is apt to be sanious and offensive, though this condition may also follow less severe lesions and be brought about by the passage from the lumen of the bowel of certain anaerobic bacteria. In such cases the peritoneum itself is likely to be necrosed. When the peritoneal exudation is of this putrid character, gas is often

present, and this without perforation of the intestines—which event, of course, gives rise to this condition. To similar anaerobic microbes the presence of the gas is then due. A feculent odour may be communicated to the exudation, especially when it is encysted, by transmission of gases from the intestinal canal without direct communication. A remarkable case in which wax-like masses composed of fat and fibrin were found in the exudation has been reported (Porak and Durante). After death it is usual to find that the exudation has gravitated into the most dependent parts, especially into the pelvis; but during life it is somewhat more distributed among the coils of intestines and among the other viscera, being kept so distributed by the movements of the bowels. Sir F. Treves, from experience at operations, found the greater portion of the fluid in the loins. When the amount is very excessive, the intestines are displaced, and oftener over to the right side. Consistent with the view that definite communications existed between the peritoneal cavity and the subjacent lymphatics—stomata—chiefly over the diaphragmatic surface and the omentum, it has been assumed that the accumulation of fluid was in great measure dependent upon the blockage of these apertures by the fibrinous exudate. But since these apertures have been recognised to be artifacts (Muscatello, MacCallum), the effusion must be attributed to some alteration in the permeability of the serous membrane, or other structures intervening between the blood and lymph currents and the abdominal cavity, which diminishes the normal absorbent capacity of the peritoneum.

Should the patient recover or live sufficiently long, the whole of the inflammatory products, fibrinous or fluid, may be completely absorbed, that is if the endothelium have suffered but little or no injury; or the fibrinous exudate may become organised into fibrous adhesions, varying in their firmness and extent from merely delicate threads to membranous films. If the effusion have been haemorrhagic, some pigmentation of the peritoneal tissues may long remain. An exception to the total disappearance must be made in the case of purulent exudations, which, undergoing inspissation and fatty change, may leave caseous masses. Very frequently, however, the pus burrows in different directions, sometimes into the intestine or bladder or through the abdominal wall, preferably at or in the neighbourhood of the umbilicus, forming an intractable fistula. This last issue specially occurs in children when the infection is pneumococcal, but it also happens in puerperal peritonitis. These modes of escape of the pus from the peritoneal cavity may take place when the exudate is free in the serous sac, as well as in the more frequent condition of an encysted abscess.

In the form of the disease described as "progressive fibrino-purulent" there are numerous foci successively developed from a primary inflammatory area, and form small collections of purulent or sero-purulent exudation more or less confined by adhesions, between coils of intestine, or between the intestines and the parietes. The firmness of the adhesions, which are frequently pigmented, varies with the duration of the disease.

There remains to be mentioned that somewhat rare condition known

as "polyserositis" or "polyorrhomenitis," in which several of the serous membranes are successively the seats of inflammation, one of them being the peritoneum. The pericardium and pleura are frequently affected as the result of several forms of infection, especially the rheumatic, though this seldom, if ever, affects the peritoneum. Excluding tuberculosis, which more often leads to a subacute or chronic affection of the several serous membranes, there are still a few cases in which the peritoneum, either before or after the pleura, is the subject of pneumococcal or streptococcal invasion. In such the post-mortem appearances conform to what has been described, with the addition of similar morbid changes in the pleura or pericardium, or in both.

Symptomatology. — Although the cause of acute peritonitis is uniformly bacterial infection, without which the disease does not arise, there nevertheless follows a great variety in the range of resulting symptoms, both in their individual intensity and prominence, as well as in the general condition of the patient. These differences may be referred to one or other of the following circumstances, or, it may be, to combinations of them:—(1) The special form of infection; (2) the mode of infection; and (3) the personal factor represented by the age and condition of the individual.

(1) That the range and severity of the symptoms are largely dependent on the special form of microbic infection has become clearly demonstrated, and to some extent the various organisms met with in association with acute peritonitis may be ranged in order according to the violence of their effects. Thus, those cases which, from the overwhelming suddenness and severity of their onset and course have been termed fulminant, invariably yield cultures of *Streptococcus pyogenes*, whilst a white staphylococcus is connected with the mildest cases. The gonococcus, when a pure infection, also appears to determine symptoms of a moderate character, whilst the pneumococcus, which is responsible for a large proportion of the acute peritonitides occurring in children, especially girls, is far more serious in its effects. Of the *Bacillus coli* there are several strains that differ in their degree of pathogenetic virulence. "The colon bacillus," say Messrs. Dudgeon and Sargent, "appears to be less virulent than the *Bacillus pyocyaneus* when present in the peritoneal cavity in peritonitis, and is certainly much less virulent than the *Streptococcus pyogenes*. If present alone in the peritoneal exudate 'at a distance,' then the prognosis is extremely grave, whilst if the phagocytes are all degenerated, then the prognosis is practically hopeless. If this organism is present together with the *Staphylococcus albus*, then the prognosis is less grave, although still very bad; if, however, the *Staphylococcus albus* has been present in the peritoneal exudate some hours before the colon bacillus, then a patient suffering from diffuse peritonitis has a far better chance of recovery."

(2) The mode by which infection of the peritoneal cavity takes place influences the occurrence of peritonitis both as regards its intensity and also in respect to its general or localised character, and this it does in

great measure by determining the amount of the infective agent and of the chemical and mechanical damage, as well as the rate by which the infection is brought about. Thus, the sudden perforation or rupture of the stomach or intestine admits at once a quantity of the noxious material far beyond what the serous membrane can deal with, and the effects in consequence are extensive and overwhelming; whilst should the infection take place gradually through intestines or appendix that exhibit no obvious break of continuity, the results are more apt to be localised, at least at first, and of less serious character.

(3) The condition of the individual attacked likewise has its effect on the development as on the course of the disease. Such circumstances as age and sex, previous health, states of debility from alcoholism, drug habits, existing maladies such as Bright's disease—10 per cent of the fatal cases of which are complicated with acute peritonitis,—as well as the normal states of menstruation and parturition, all have an influence by diminishing or modifying the resisting power of the peritoneum. Acute peritonitis from all causes is of most frequent occurrence between the ages of ten and forty, infection by appendicitis being chiefly responsible for the younger cases, as the puerperal state and strangulated hernia account mainly for the later. The great frequency with which the disease starts from the reproductive organs in females is probably nearly balanced by the far greater preponderance of appendicitis among males.

These several etiological factors have already been shewn to be chiefly responsible for the differences in anatomical characters manifested in peritonitis, such as its generalised or local distribution, as well as the nature of the inflammatory products, serous, fibrinous, haemorrhagic, suppurative, or adhesive; so also are they connected with the clinical varieties of the disease, whether acute, subacute, or chronic, and with the preponderance of individual symptoms referable to the seat of the mischief, as well as those of a general toxaemic nature. Hence there come to be—within the limits of acute diffuse peritonitis—certain clinical forms characterised by greater or less prominence of certain features, as there are also numerous cases that may be regarded as occupying intermediate positions. No single description can be furnished that will include all cases, and it will be more convenient, therefore, to give so far as possible a word-picture of the more marked forms, and afterwards to discuss *seriatim* the various symptoms that may occur.

First may be described a case of acute peritonitis caused by the perforation of appendix, stomach, or intestines in an individual who, up to the occurrence of the lesion, presented no symptoms of illness, or nothing beyond a very slight discomfort. It may be that such a patient, when going about his ordinary avocations, or exerting himself in some manner a little out of the common, is suddenly seized with severe abdominal pain, sufficient, it might be, to cause him to scream out, accompanied possibly with a sensation of "something having given way" in the belly, and rapidly followed by a feeling of faintness and collapse. These

phenomena, differing but slightly in their degree, may be said to characterise all forms of perforation of the hollow viscera which contain air, and, with a greater variation of intensity, are met with in rupture of the gall-bladder, of the urinary bladder, of the solid viscera, or the bursting of an abscess or an empyema into the peritoneal cavity. It sometimes happens that the patient is able to refer the pain at its immediate onset to the epigastric or caecal region or elsewhere, but it rapidly becomes spread over the whole abdomen, and no recollection or indeed observation of its starting-point may be retained. The aspect of the patient betokens great anxiety and distress, the features are pinched and drawn, and the countenance pale, and bedewed sometimes with a clammy sweat. A feeling of nausea is experienced, and there may even be vomiting. The pulse is small and rapid, and the respirations are quickened and shallow. Should there be any very great escape of gas from the perforated viscus into the serous cavity, the abdomen will be distended and tympanitic, with possibly a disappearance of the liver dulness, though this is far from constant and by no means diagnostic; otherwise the abdomen is likely to be retracted and rigid. The surface temperature is likely to have fallen somewhat, but occasionally there may be a rise and even a rigor, though this is exceptional.[1] So far the symptoms are those of perforation only, and these are fully discussed elsewhere (p. 474). It may be that the degree of shock or collapse induced by the lesion is so profound that the patient never rallies, and dies within a few hours, although this is of very rare occurrence in a patient who has previously been to all intents and purposes in good health, such as is now assumed to be the case. As yet the evidences of peritonitis have not appeared, and it will be from four to eight, or even twelve, hours before they do, the initial symptoms of perforation either merging into those determined by the inflammation of the serous membrane in such a way that no line of demarcation can be drawn, or, as is not infrequent, with some recovery from the shock and abatement of the other phenomena—a "period of repose," as Mr. Symonds described it — previous to the onset of the peritonitic manifestations. The pain continues to be the prominent feature, amounting in severity to what may well be described as agonising, absolutely continuous and persistent, though marked now and again by exacerbations, and intensified by every movement that involves the abdomen or its contents; even emptying the bladder is sufficient to evoke a paroxysm. To avert so far as possible an increase in the suffering from this cause, the patient lies with legs drawn up and arms raised above the head, thus relaxing the parietes and giving a better opportunity for action of the extraordinary muscles of respiration, whereby compensation is made for the inactive diaphragm, the movements of which greatly add to the pain. Hence the breathing is almost wholly costal in character, becoming, as

[1] A certain group of symptoms, chief among which are shock, collapse, vomiting, and pain, which are common to most acute abdominal attacks, whatever may be the viscus chiefly concerned, but in which the peritoneum is involved has been denominated "peritonism."

the disease progresses, more and more hurried and shallow, reaching 40 or even 60 per minute. Yet, though every effort is instinctively directed towards keeping the abdomen absolutely still, the patient is nevertheless possessed by a disturbing restlessness that persists often to the very end; and piteous is it to see him toss his head from side to side, now and then in a moment of forgetfulness giving a turn to limb or trunk that causes a deeper twinge of suffering. It is this association of agonising pain with an ill-restrained restlessness that gives to the severest cases their most characteristic and at the same time pathetic aspect. Should the abdomen have escaped the condition of peritoneal meteorism, the walls continue rigid, possibly retracted and quite motionless, exquisitely tender to pressure, and usually unable to bear the weight of the bed-clothes. As the intestines become paralysed and distended with gas, the parietes yield, causing the belly to be tense and tympanitic, with a variable extent of dulness forwards from the flanks according to the degree of inflammatory exudation. Whilst there is absolute constipation, occasionally relieved in the latest stages, vomiting sets in early, and persists with distressing frequency, adding greatly to the patient's suffering; small in quantity, the ejecta soon become most ill-smelling, though rarely faecal. The tongue, at first dirty with a thick white coating, later appears dry and brown, while sordes cover the teeth and angles of the mouth, and the breath is extremely offensive. Great variability exists as regards the temperature, though it is commonly, but not always, raised, in the rectum if not in the axilla, at least at some time in the course of the disease. A constant thirst racks the patient; this is ill-relieved by ice or sips of hot water, that oftener tend to excite the vomiting and increase the misery. Very characteristic is the pulse, which from the first is small, rapid, and wiry, increasing in rate and diminishing in volume and tension as the case progresses, until finally it can scarcely, if at all, be counted. The urine is scanty almost to suppression, and may contain a trace of albumin, but is especially characterised by the presence of a large amount of indican. Meanwhile the collapse and prostration progress slowly but surely, the deeply anxious expression, the drawn and pinched features, the sunken eyes with dark surrounding rings, the ashy countenance, gradually passing to cyanotic as death approaches, making up a characteristic picture of the *facies hippocratica* that from the earliest times has been associated with the gravest abdominal maladies. Add to this the cold extremities, the growing voicelessness, the never-ceasing restlessness, and the scarcely impaired consciousness, with possibly some abatement of the pain and tenderness and perhaps of the vomiting as the end draws nigh, and it is difficult to conceive a more painful condition to watch, made even more painful by a knowledge of how impossible it is to avert. "However distressing the aspect of the patient may appear, there are reasons to believe that death from acute peritonitis is not so full of suffering as might be supposed" (Treves). Many of the characteristic appearances, such as the rapidly-developed shrinking of the body, the pinched features, sunken eyes, parched mouth, and constant thirst, are

due to the great lack of fluid in the tissues from the small intake and increased loss brought about by the vomiting.

Such are the course and symptoms of a *fulminant* case, lasting on an average from twenty-four to thirty-six hours from the commencement, seldom longer, sometimes terminating sooner. Although certain of the symptoms are peculiarly referable to the peritoneal inflammation, the whole clinical picture, in which the profound shock and collapse stand out prominently, suggests that the patient's condition is one of violent poisoning rather than such as may be connected with a comparatively speaking localised affection, and it is to the general state more than to any abdominal change that the almost invariably fatal result is to be ascribed.

In marked contrast to the foregoing are those cases of acute peritonitis which, from the paucity and obscurity of their symptoms, may be termed *latent* or *masked*. No better description can be given of this form of disease than the following by Sir F. Treves in the previous edition of this work :—"Peritonitis may run its course and end in death, and yet the symptoms be but very faintly marked, or be equivocal. This is not uncommon in the old and broken-down. Such patients soon sink into an apathetic, 'typhoid' condition, complaining of little or no pain, and scarcely troubled with vomiting, while motions may be passed unconsciously in bed. I have seen such patients pat the abdomen to shew that there is no tenderness in the region. Meteorism in such cases is seldom absent, and the rapidity with which the patient breaks up is often remarkable. The tongue soon becomes dry, the pulse is uncountable, the extremities become cold, and signs of death become rapidly evident. In the subjects of Bright's disease, and in characteristic patients who are saturated with morphia, a like obliteration of symptoms may be noted ; the same also may be seen in peritonitis concurring with pyaemia. Now and then I have seen the same masked form of peritonitis follow upon operation in patients who were under middle-age, and who were constitutionally healthy. In two of these instances little or no pain was complained of, and little morphia was called for ; the vomiting was quite insignificant, tenderness of the belly was absent, and meteorism was scarcely perceptible. The one predominating symptom was a never-ceasing and extravagant restlessness which appeared to wear the patient out, and which ceased only with death about the sixth day. In a third case there was slight fever and very marked meteorism, but no pain and no vomiting. The tongue kept suspiciously foul. Food was well taken and the bowels acted well. Unceasing restlessness was the most marked feature. The pulse failed, increasing in rapidity on the sixth day ; death took place on the seventh day."

Should peritonitis supervene upon enteric fever, whether with or without perforation, the symptoms are likely to be very much in abeyance notwithstanding that the anatomical changes may be very well marked and extensive. An extreme degree of the "typhoid state," when the patient lies apathetic with blunted sensibility and in a condition of profound

asthenia, would go far to account for this, though not altogether, for the same mild character of the clinical features of peritonitis has been noticed when the complication has occurred in patients exhibiting only the usual symptoms of a mild attack of enteric fever without perforation, though the peritoneal inflammation has been intense (Goodall). Similarly, when peritonitis has followed on perforation from malignant disease of the stomach or bowel with extreme cachexia, or when it has occurred as a terminal phase of chronic Bright's disease or other grave constitutional malady, the evidences of its existence may be so slight as almost to escape notice until post-mortem examination; and when the disease develops in the new-born, it can hardly be said to be manifest during life.

Between these extremes most of the cases are to be found differing greatly in the mode of onset, in the course and severity of the several symptoms, which in many are chiefly local in character, as in others they are overshadowed by the general features of a septic intoxication, and also in the degree of fatality that marks their issue.

Having regard to the wide range of clinical characters, the diverse modes of origin, the different anatomical appearances, and in some measure the variety of the infection, that are presented by the numerous cases comprised within the term "acute diffuse peritonitis," it is found convenient to arrange them artificially in several groups, neither the limits nor the distinctive characters of which, it must be admitted, are well defined. Such are (i.) Perforative peritonitis, (ii.) Peritonitis of appendicular origin, (iii.) Pneumococcal peritonitis, (iv.) Gonococcal peritonitis, and (v.) Puerperal peritonitis. It is likely that with further knowledge this arrangement will be modified and extended.

(i.) **Perforative Peritonitis.**—This has already been described, and it is sufficient to add that when the perforation of the viscus takes place very slowly, or the aperture is exceedingly small, the morbid changes may be localised, or if they become general, do so somewhat more gradually than when the lesion is considerable in extent and of sudden occurrence.

(ii.) **Diffuse Peritonitis of Appendicular Origin.**—Inasmuch as mischief in the appendix is the starting-point of the greater number of cases of acute diffuse peritonitis, this circumstance alone suggests the convenience of grouping them together. Not that they exhibit any specially distinguishing clinical features, which indeed conform to what has been already described, or will be in the following sections, as characteristic of acute peritonitis generally. There is little doubt but that most cases of general peritonitis originating from the appendix develop in the first attack of appendicitis, and it is this which determines the high mortality of the latter affection. The morbid conditions of the appendix which are responsible for setting up the diffuse peritoneal inflammation are (*a*) gangrene and perforation, which are the cause of most of the fulminant cases and are referable to the previous group; (*b*) purulent inflammation, without actual perforation (empyema of the appendix), which may be of a

sufficiently virulent and extensive nature to lead to the invasion of the whole serous cavity; and (*c*) the gradual extension to the rest of the peritoneum from what was at first an encysted or localised inflammation (perityphlitis). The last variety, also known as "progressive fibrino-purulent peritonitis," is characterised by a spreading of the morbid process from where the adhesions and the fibrinous deposits at first confined its effects. Considerable differences will exist as to how far the extension proceeds, influenced in great measure by the duration of life of the patient, but occasionally the whole peritoneal cavity may become affected, there being found collections of purulent or sero-purulent exudation imperfectly confined by fibrous bands among the coils of intestines and between the other viscera. A similar extension may take place from a peritonitic abscess that has developed elsewhere than the caecal region, such as around the kidney, beneath the diaphragm, or in connexion with a mesenteric or retroperitoneal gland. Clinically such cases exhibit a progressive character in the development of symptoms, and the condition may go on for weeks. If watched from the commencement, the pain will be recognised as extending from the region of origin and in time to become general, but its acuteness and severity, the associated tenderness as well as the rigidity of the abdomen, or the marked constipation and meteorism which are so prominent in perforative peritonitis are much less prominent, or may be in great part absent. Indicating fresh accumulations of pus as the mischief spreads, the temperature exhibits a curve characterised by periodic remissions or complete intermissions, rigors and sweats accompanying each successive rise (*vide* also p. 606).

(iii.) **Pneumococcal Peritonitis.**—Acute diffuse peritonitis due to infection by the *Pneumococcus* comprises a group which is characterised by fairly distinctive appearances. The fullest account of this variety of peritoneal inflammation is that furnished by Dr. Annand and Mr. Bowen, who have collected and analysed the records of ninety-one cases in children under fifteen years of age, some of them being but a few weeks old, and one at least only a few days. This age-incidence is itself remarkable, for comparatively few cases were met with among adults. Scarcely less singular is the great preponderance of females, who formed 73 per cent of the total cases. They were about equally divided into diffuse and circumscribed, and whilst in twenty-one of the whole number the peritoneum appeared to be the primary seat of the mischief, which was general, nineteen of the diffuse cases were secondary to, or connected with, a similar inflammation in other parts. The source of the infection of the primary cases was chiefly the intestine, a few starting from the appendix, which appeared to be the more frequent origin among the few adult patients. Catarrhal states of the gastro-intestinal mucosa were the usual morbid changes found, and this apparently was sufficient to permit the invasion of the peritoneum through the damaged gut by the pneumococcus, which is an occasional inhabitant of the alimentary canal. The great frequency of the disease among female children suggested the genitalia as a probable channel of infection, but no pathological support

for this view can be adduced. But far the greater number of the secondary cases were associated with pneumonia, bronchopneumonia, or empyema, some of which were further complicated with pericarditis. In a few cases the primary affection was otitis media, sore throat, or intermuscular abscess, and the invasion of the peritoneum could in these be only by the blood-stream. Probably also it was by the same means that the pulmonary cases gave rise to the peritonitis, rather than by spreading through the diaphragmatic lymphatics, and though this route is prima facie probable, much may be said against it, not the least objection being that such an invasion would be against the lymph-stream. In respect to the symptoms exhibited by these cases, whether primary or secondary, they were always severe, closely resembling, with one exception, the clinical course of diffuse perforative peritonitis. There was the same sudden onset, often with rigors, violent and persistent pain, frequent vomiting, advancing prostration, and early death, but in place of constipation there was a considerable diarrhoea, the expression alike of the causal enteritis and of the profound septicaemia. It was noticeable also that in many of the secondary cases the peritonitic symptoms were considerably or even completely overshadowed by the general toxaemic state, or by those connected with the other seats of the pneumococcal invasion. On the whole, there is a good deal of resemblance in the symptoms and general condition which are associated with pneumococcal invasion of the lung to those of a like infection of the peritoneum, and "further it is said if the patient live long enough the temperature will fall quickly about the seventh day as it does in pneumonia" (Hale White). Other observers have pointed out that the inflammatory exudate, which is of a greenish odourless purulent character, has, if not removed by operation, a marked tendency to escape at the umbilicus.

(iv.) **Gonococcal Peritonitis.** — Although diffuse gonorrhoeal peritonitis is of a much more frequent occurrence in females, in whom a clear and open channel exists for the conveyance of infection to the serous cavity, it is not altogether unknown in men as the sequence of gonorrhoeal epididymitis. Battez collected the records of thirty cases in males, in some only of which was the inflammation diffuse, others being localised in the pelvic region. Commonly, no doubt, it is a mixed infection, but pure cultures from the peritoneal exudation have been obtained (8, 9). Although the usual symptoms of acute peritonitis are generally well marked and present nothing distinctive, the outlook is commonly favourable both in men and women.

The condition has been met with among girls of from five to twelve years of age, the gonococcus being found in the vaginal discharge and in the peritoneal exudate when the abdomen was opened. Of eight cases collected by Comby all recovered without operation, though the symptoms in several were severe. Cases fatal after laparotomy have been recorded by Hunner. Considering the frequency with which acute peritonitis starts from the vermiform appendix in children, the importance of thorough examination is obvious, lest the true source be overlooked.

(v.) **Puerperal Peritonitis.**—The peculiar conditions under which this form of peritoneal inflammation takes place, as well as certain characteristic appearances, fully justify its relegation to a special group. Originating through septic absorption from the uterine surface at or after delivery, and almost invariably due to the *Streptococcus pyogenes*, the symptoms set in usually on the second day after confinement, occasionally sooner, or they may be postponed for several days later. Absorption chiefly takes place by the damaged lymphatic vessels, and so may spread through the walls of the uterus to the peritoneal surface, or the septic material may find its way through the Fallopian tube into the serous cavity. More frequent in primiparae, in whom injury of the passages during parturition is likely to be greater, the retention within the uterus of portions of placenta or blood-clots undergoing putrefaction is especially contributory to its occurrence. Although the puerperal variety conforms in all its main features to the general type of the disease, it is more particularly distinguished by the predominance of symptoms referable to septic intoxication, and tends towards a fatal termination somewhat sooner than other forms of the malady, except those following on visceral perforation, death usually taking place within a week. It is this strongly developed septicaemic character that explains the greater frequency with which diarrhoea occurs, although marked constipation is far from unknown. Chills and rigors not infrequently indicate its onset, and the temperature generally ranges high. The pain, which is severe, usually starts from the hypogastric region, whence it spreads over the whole abdomen, which, owing to the laxity of the parietes following on labour, soon becomes enormously distended from intestinal gases, the more characteristic rigidity being much less obvious. It only remains to add that the uterine discharges are from the first very offensive and the milk secretion is suppressed.

It is desirable to consider a little more in detail some of the signs and symptoms already mentioned, distinguishing between those which are referable more particularly to the peritoneum and involved viscera that may be looked upon as local, from those which are to be regarded as manifestations of the induced toxaemia as affecting either the body generally or organs outside the abdomen. It is necessary also to discriminate between those phenomena set up by the peritonitis and those of the primary disease of which it is a complication.

Signs and Symptoms of Peritonitis in Detail.—*Pain and Tenderness.*[1]—Except when the symptoms are masked, a continuous agonising pain is an

[1] The *sensibility* of the peritoneum is a very difficult subject about which much difference of opinion exists and much remains to be explained. Lennander (14), who has made numerous observations during abdominal operations under local anaesthesia, is strongly of opinion that the parietal layer of the serous membrane, that is to say the portion overlying structures supplied by somatic nerves only, is alone sensible to painful impressions, whilst the visceral layer is quite insensitive to similar stimuli and, like the entire peritoneum, to

invariable accompaniment of acute peritonitis, and constitutes one of its most distinguishing features. The chief characteristics of the pain are its excruciating severity and steady persistence, marked though this may be by occasional exacerbations attributable to no very obvious cause unless to movements of the intestines. When the cause determining the disease is of sudden onset, as a perforation of appendix or stomach, the acute pain attendant on the lesion may continue into that set up by the peritoneal inflammation, and very possibly with little or no remission to mark the difference; when the primary mischief is of slower development, the onset of the peritonitic pain is more gradual, though it is seldom long before it attains to a degree of intensity that equals that of the more rapidly established cases. Whilst in its general distribution it tends to spread over the whole abdomen, and even to extend to the upper part of the trunk, it is not infrequently of somewhat greater acuteness in certain spots, such as the epigastric, caecal, or hypogastric regions, which may correspond to the site of the initial lesion, though with sufficient infrequency to render the indication unreliable, or, as is so common in intestinal diseases attended by pain, is more specially referred to the region of the umbilicus. Without doubt the severity and extent of the pain are in great measure proportionate to the suddenness of the attack and the area of peritoneum exposed to the causal irritant, and as a rule these conditions best obtain in the perforative form of the disease; whilst in the peritonitis of puerperal origin the pain may be much less severe. Two circumstances are likely to be attended with some abatement or even complete subsidence of the pain: a considerable inflammatory exudation by which the inflamed surfaces of the membrane are somewhat protected and kept from rubbing on each other; and intestinal meteorism, which is also often associated with a diminution in the pain, due perhaps in part to the paresis of the intestines, and partly to the general asthenia of which this may be an expression. In cases in which the toxaemia is profound and a semi-comatose condition is established, the pain almost or quite disappears, and the patient, though rapidly drifting to death, may appear quiet and comfortable. Some difference in the degree of suffering experienced by different patients may be attributable to personal

heat and cold; and that this is equally true whether the serous membrane be healthy or diseased. It cannot be said that these views are universally accepted, but the evidence Lennander adduces strongly supports his contention that it is only when traction forwards is exerted on the omentum or mesenteries, or when the parietal surface is manipulated or rubbed, that any pain is evoked. He even explains the pain of intestinal colic, or that perceived in any of the viscera, as due either to a distension of them which causes dragging on the peritoneum, or to mechanical or toxic means which irritate the terminal branches of the lumbar or sacral nerves in the parietal subserosa; even lymphangitis and swollen lymphatic glands secondary to inflammatory or other morbid states of the peritoneum or viscera he regards as acting in the same manner. There is certainly much to be said for this view if only because the abdominal viscera generally are remarkably insensitive to handling, or even stronger measures such as cutting and burning. So far as it goes also, it is supported by the current doctrine regarding the innervation of the structures concerned. Prof. Langley considers that those fibres which on stimulation are capable of giving rise to sensation belong to the somatic system, of which variety the lumbar and sacral nerves from the spinal cord mainly consist, whilst there are far fewer such fibres in the sympathetic branches distributed to the viscera.

idiosyncrasy and the tolerance or otherwise of pain, but when every allowance is made on this point acute diffuse peritonitis remains one of the most exquisitely painful of all diseases.

Scarcely less marked than the spontaneous pain is the tenderness on pressure, that may be so acute as to cause the patient to wince at the mere approach to being touched. Some degree of pressure, however, is usually required to elicit this symptom slight though it be, for the skin alone may sometimes be pinched without discomfort, provided there be no disturbance of the underlying structures involving the parietal peritoneum. As with the pain, so the tenderness may be first manifested in the region of the causal lesion, though it quickly tends to become general, and may even extend round into the loins.

Condition of the Abdominal Walls.—Closely connected with the pain, and, like it, a direct result of the peritoneal irritation, is the appearance presented by the abdominal muscles. Quite one of the earliest signs is the rigidity and immobility of these structures, in the nature, as it were, of a reflex defensive action to protect the inflamed peritoneum from pressure and further injury. Varying degrees of this resistant hardness of the parietes are met with, but it is in perforative peritonitis, more particularly when the lesion is gastric, that the most extreme states occur. In such the contraction may be so great, and accompanied with similar spasm of the intestines, as to cause a retraction of the front of the abdomen almost on to the spine, "comparable to that sometimes met with in lead colic" (Treves). In cases which are not of perforative origin and come on less suddenly, the degree of retraction is less, but the muscular rigidity is always early in appearing. In the most rapidly fatal cases death takes place whilst the abdomen remains rigidly retracted, but it is usual that some relaxation takes place after a time, and in the later stages the belly is distended and tympanitic from accumulation of gas in the paralysed intestines. Where the asthenia is very considerable the muscular rigidity is insignificant, and Sir F. Treves records having "seen cases of septic peritonitis run their entire course with a flaccid belly wall, and with scarcely any tenderness. In such instances the patient is usually past middle life."

The later distension of the abdomen may also be due in part to the fluid *exudation* into the peritoneal cavity. The amount of this varies within very wide limits, but it may be sufficient to give rise to all the physical signs of a considerable ascites. The free mobility of the fluid within the serous sac is apt to be interfered with to a greater or less extent by adhesions, which tend to confine the exudation unequally in different regions of the abdomen, thus modifying the characteristic limits of dulness and resonance as met with in dropsy of cardiac or hepatic origin. Now and then a friction fremitus may be both felt and heard, chiefly over the upper zone of the abdomen, caused by the rubbing together of the roughened surfaces of the peritoneum, as the organs and parietes move with respiration, or even from the intestinal peristalsis. In exceptional cases there may be some oedema of the integuments over

the abdomen when the inflammatory exudation is purulent in character, comparable to the condition which may be observed over the chest in connexion with an empyema.

Vomiting.—This, another of the characteristic symptoms of acute peritonitis, is also at first and often throughout a reflex phenomenon excited by irritation of the peritoneum. It is singularly persistent, appearing at the very outset and continuing sometimes until death occurs, and it is only with its cessation that any improvement in the patient's state can be looked for. The more acute the case, and especially if perforation have happened, the more marked the vomiting, though should the stomach be empty when the lesion occurs it may be but slight and is said not to occur when the gastric tear takes place into the lesser peritoneal sac. Notwithstanding that it is one of the chief manifestations of peritoneal inflammation, the extent to which it is met with is variable when the different causes of peritonitis are taken into consideration. It has been also noted that when the bowels are acting regularly or when there is diarrhoea—as may be the case when peritonitis supervenes on enteric fever — the vomiting is likely to be inconsiderable. Inasmuch as a looseness of the bowels is not infrequent in the puerperal form of peritonitis, vomiting is often less marked in this class of case. When the peritoneal exudation is very considerable, the sickness is said to be less. Since the emesis may frequently be excited—or at least appear to be—by the taking of fluid, however little or of whatever character, it would seem that the act may be reflexly excited from the gastric mucous membrane as well as by the peritoneal inflammation. The vomited matters are at first such material as the stomach may happen to contain, later becoming bilious in character, then green from altered bile, and later brownish and very offensive, though seldom actually feculent. When the peritonitis is associated with grave toxaemia blood may be found in the vomit (5). Unless the peritonitis complicate acute intestinal occlusion, when the amount of the ejecta may be copious, the quantity is usually small and brought up with but little effort, giving no relief to the patient, but on the contrary greatly intensifying the pain. In the later stages of the disease the vomiting may be replaced by simple retching, nothing being brought up until perhaps at the moment of death a final ejection of feculent fluid may take place. Eructations of ill-smelling gas are not infrequent. Mere nausea apart from the vomiting is less often complained of.

Hiccup from spasmodic contraction of the diaphragm is yet another reflex manifestation of peritoneal irritation, which is of occasional occurrence and adds greatly to the patient's distress.

Condition of the Intestines.—The intimate connexion of the peritoneal covering with the other structures of the bowel wall admits of a ready extension of the results of the inflammation of the serosa to the muscular and other coats. It is clear that these changes, apart from any interference with the nervous control of the musculature, would be likely to inhibit peristalsis very considerably, and hence it is that in the majority

of cases of acute diffuse peritonitis from all causes, *constipation* is met with. Nevertheless in a certain proportion of cases there is no evidence of functional impairment of the mobility of the intestine, and the bowels act regularly or almost so, or do so with but very slight assistance. Although the more usual condition of constipation, which may for the time be absolute and resist all treatment, may be accounted for in the manner described, at least in the later stages, it is probably dependent in a measure on reflex inhibition of the nervous control by the primary lesion, such as perforation or acute occlusion of the intestine, or by the irritation of the peritoneum. This explains the early onset of constipation before indeed the structural inflammatory changes in the bowel can have become established. Even though the constipation be complete, there may be an occasional passage of flatus *per anum*.

In a comparatively small number of cases of acute diffuse peritonitis *diarrhoea* has been observed, more particularly among those in which the disease originates in the pelvis, especially the puerperal form, and in the pneumococcic peritonitis of children; sometimes also in the perforative peritonitis complicating enteric fever. This exceptional condition is regarded as a direct result of the septicaemia, the toxins of the infection being absorbed and possessing an unduly stimulating effect on the neuro-muscular mechanism of the bowel, comparable to the corresponding irritation of the neuro-secreting mechanism by the toxin of cholera.

A most important result of the intestinal paralysis is *meteorism*, due to an accumulation of gases within the relaxed and yielding bowel. First observed over the upper zone of the abdomen, it spreads and may attain enormous dimensions, stretching the integuments to their utmost limits and making them tense, smooth, and shining. To such an extent may this condition reach that the movements of the lower part of the chest are completely arrested and the heart displaced upwards, with diminution of the areas of the hepatic and splenic dulness. Some of the distension may be caused by the fluid exudation, but this alone never determines the extreme girth that meteorism may induce. It is obvious that for any distension to occur, the rigidity of the abdominal muscles must have subsided and been replaced by a more or less flaccid condition, and this loss of tone of the somatic systemic muscles is to be regarded, like the intestinal paresis, as an outcome of the septicaemic state, and is consequently of proportionately grave significance, which is increased with the persistence of its duration. For these reasons meteorism seldom appears within the first twenty-four hours of the disease, and is usually manifest in the course of the second day, being very variable in extent and sometimes absent throughout: such circumstances as parturition or previous ascites, which have unduly increased the laxity of the parietes, favour the distension, which may then become enormous.

Among other muscular paralyses that should be noticed is that of the bladder, which may become invaded from the serous coat of this organ, as the intestinal muscles are, so causing inability to pass urine. In contrast to this a frequency of micturition is sometimes noticed.

General Condition.—Although acute diffuse peritonitis is primarily a local disease characterised by the features just discussed, yet owing to its infective nature it sooner or later gives rise to general symptoms determined by toxic absorption from the peritoneal cavity. Hence the disease is in all cases a toxaemic disease, differing as it may in the degree of the toxic phenomena. Excluding those somewhat exceptional cases which are fatal in a few hours subsequent to perforation of stomach or bowel, the patient never rallying from the collapse attendant on the visceral lesion and dying before peritonitis has actually commenced, there remains the whole field of acute diffuse peritoneal inflammations, ranging from those of a fulminant character, of which a description has been given, to those which linger on for weeks, whether ultimately recovering or not. Within that wide category the extent to which the general indications of toxaemia are manifested is extremely variable. In the severest cases, those in which it may be that the initial collapse is followed by a "period of repose," and this is succeeded by the supervention of the local signs of peritoneal inflammation, the evidences of intoxication rapidly become dominant, and it is from poisoning that the patient dies. That this is so, the general aspect and expression, the pulse and temperature, the oftentimes icteric tint so significant of serious blood change, and the not infrequent mental disturbance sufficiently declare. But although the characteristic appearance and abdominal facies are most usual, they are by no means invariable even in cases that are surely drifting to their death, and an expression that but very slightly betokens the serious condition, with even a flushed countenance, has been observed within a few hours of the end.

Where the infecting organism is of a lower grade of virulence, or the amount of toxic matter that obtains entrance to the peritoneal cavity is but slight, or the associated chemical and mechanical damage to the peritoneum by the escaped gastric or intestinal contents is insignificant, or conceivably when the absorption of toxins into the blood-stream is but small, then the probabilities are that the general evidences of septicaemia are moderate, and the outlook for the patient is proportionately better as the more particularly abdominal symptoms predominate.

Thus, acute diffuse peritonitis may from its symptomatic aspect be regarded as presenting three main types of cases: those in which the general toxic features are overwhelming and insistent from first to last; those in which the local symptoms are pronounced with a growing preponderance of the manifestations of poisoning; and, lastly, those less common and milder cases in which the abdominal signs make up the main character of the attack and the toxic effects are altogether subordinate.

Pyrexia.—Among the results of toxic absorption is the disturbance brought about in the heat-regulating centres of the body, with consequent perturbations in the bodily temperature. Although of considerable interest, it cannot be said that this sign is, with our present knowledge, of much value from either a diagnostic or even a prognostic aspect, and since the range and course of the pyrexia vary so exceedingly, no

approach to a characteristic curve having any general application can be constructed. That there should be this variability is to be expected when account is taken of the diverse conditions that occupy a primary position in the etiology of the disease, even if the numerous localised and chronic forms be disregarded and only the acute diffuse inflammation be considered. Nor are these differences in cause the sole influencing circumstances, for without doubt the quantity and virulence of the infection, and probably also the species of organism, affect the character of the temperature, as well as the appearance of certain symptoms which always depress the temperature during the progress of the peritonitis, such as diarrhoea. No special connexion between the nature of the inflammatory exudation and the degree of fever is to be observed, for pus may be formed without any special rise and even sometimes with a subnormal temperature; on the other hand, the highest readings are commonly reached in cases associated with suppuration, and in purulent peritonitis left untreated for some time the temperature is marked by daily remissions and intermissions of considerable range, the so-called hectic fever.

In the majority of all cases of acute diffuse peritonitis the temperature at some time or other and for a variable period is raised; the maximum attained rarely exceeds 104° F., and more often does not go above 103° F. Perforation is usually attended with a fall of the thermometer, and should the temperature at the time be raised as in enteric fever, this fall is obviously more marked; but occasionally the fall does not take place, and a rise has even been known to accompany this event. In some cases the collapse attendant on this lesion may be so profound that death may ensue without any subsequent rise, before, that is, peritonitis is established; but oftenest with the onset of the inflammation, if not before, the temperature rises and quickly reaches its highest point, at which it may remain for some hours or for a few days, according to the course of the disease, with daily irregular fluctuations, later falling even to subnormal and so remaining until death, or this event may be immediately preceded by a sudden rise to 103 or 104 F. If the peritonitis be of slower onset, the temperature rises more gradually, but as before exhibits no constant types. In a small proportion of cases, among which may be included the acute peritonitis following on strangulated hernia, the temperature remains normal or subnormal throughout, without even a rise being recorded, or it may be that after an initial rise lasting for a day or so the temperature falls to subnormal and so remains. Nothnagel remarks that the absence of pyrexia in his experience is specially frequent in cases due to injury. With respect to the low temperature, it must be remembered that very often a higher reading is obtained in the rectum, and this difference is more particularly marked when the patient is collapsed. It will thus be seen that very little assistance towards ascertaining the cause of the peritonitis can be obtained by a study of the temperature chart in a given case, any more than a forecast of the issue may be drawn from the

same. Death occurs with a high or a low temperature, or with one that remains fairly steady at, above, or below normal. Those cases marked by the highest temperatures may improve, but certain it is that those marked by a persistence of low temperature are amongst the gravest.

As previously stated, *rigors* are not a very frequent symptom, and are by no means a necessary accompaniment of pus formation. Of 100 cases quoted by Sir F. Treves from the records of the London Hospital, this symptom was only noted in 13 instances. Perhaps in the peritonitis of pneumococcal origin and the puerperal variety they are most frequent and most severe.

Symptoms referable to the Circulation.—The most important of these is the state of the pulse, which is generally characteristic, being rapid, small in volume, and hard, a combination that constitutes what is known as "wiry" or "thready." This feature is better marked in the earlier stages of peritonitis, and special attention has been drawn to the sudden rise of blood-pressure as one of the very first signs of the occurrence of perforation and consequent peritoneal inflammation in enteric fever (Briggs). The pulse-rate is usually upwards of 120, and in the final stages may reach 160, but whilst still continuing to be very small, the tone of the vessels has then diminished and the peculiar wiry feel is no longer noticeable. There is no constant relation between the pulse-rate and the temperature, for, whatever the latter may be, the former keeps high and quite contrary in character to the soft full pulse so common in the febrile state. Where the peritonitis is more slowly developed, as in some of the non-perforative forms, there is a closer correspondence between the pulse frequency and the temperature. The rate and character of the pulse are doubtless expressions of the toxaemia, although some influence may be attributed to the severe pain, acting, it would seem, on the nerves of the vessels by paralysing the vasomotor centre in the medulla oblongata (20).

The *urine* presents the usual febrile characters, being scanty and high-coloured, depositing urates and containing a trace of albumin. In addition it is characterised by the presence of a very considerable quantity of indican. "There is probably no other condition," says Nothnagel, "with the exception, possibly, of acute intestinal obstruction, in which the amount of indican in the urine is so large." The micro-organism to which the peritonitis is due may be found in the urine within twenty-four hours of the onset (Lennander (13)).

The blood in peritonitis shews a well-marked leucocytosis except in those cases of extensive perforation in which the patient succumbs in a few hours without rallying from the initial shock. Ordinarily the leucocyte count is about 20,000 per c.mm. though amounting in extreme cases to 100,000 per c.mm., being unsurpassed in this respect by any other disease. The increase is almost entirely in the polymorphonuclears.

The Mental Condition.—The patient usually remains with unimpaired consciousness to the end, even when the issue is rapidly fatal. But the degree and character of the toxaemia is sometimes such that various

conditions of mental disturbance are manifested. More commonly this takes the form of a quiet delirium or a mere confusion and obscuration of the intellect, or it may be deep drowsiness and semi-coma. Exceptionally the delirium is of a wilder character, even maniacal, and it is possible in such cases that a coincident meningitis may be responsible, especially if there be also headaches. The distressing restlessness has already been alluded to, and with it goes as a rule an equally exhausting sleeplessness. In children convulsions are not infrequent.

Notwithstanding the widespread area involved in acute diffuse peritonitis and the virulent character that the inflammation often assumes, no extension of the process takes place into the subjacent viscera covered by the membrane, beyond some oedematous and round-celled infiltration referred to in connexion with the intestine. The disease, therefore, is very rarely marked by any complication, though it may by extension through the diaphragm set up pleurisy and empyema, oftener, it is said, on the right side; but these affections, as also pneumonia, meningitis, and endocarditis, all of which are not infrequently associated with pericarditis, and arthritis, are more properly to be attributed to coincident infection, and hence it follows that it is in the more virulent cases, such, for instance, as the puerperal form, that these manifestations are most likely to occur. Rapid enlargement of the spleen has been occasionally described.

Diagnosis.—It is not often that acute general peritonitis fails to be detected unless in the exceptional circumstances that mask its manifestations, and this notwithstanding that no single feature is pathognomonic and even combinations of its symptoms may closely simulate other maladies. The difficulty in connexion with the disease lies rather in ascertaining the precise cause for the diffuse peritoneal inflammation, and this particularly so when the case is of the fulminant perforative variety.

Among the indications upon which most reliance is to be placed for the recognition of the condition are the acute pain and tenderness, the rigid motionless abdomen, the persistent vomiting and constipation, the peculiar small, rapid, thready pulse, the distressing restlessness, and the progressive prostration and collapse; yet no one of these is of invariable occurrence and it is rather on a combination of these signs that the judgment is based. Valuable confirmatory evidence is the recognition of peritoneal effusion. Some signs, such as the temperature, are so uncertain as to be of but little value, even if they be not misleading. Certain phenomena stand somewhat apart in their significance; thus, the very marked leucocytosis is eminently suggestive and becomes especially valuable should the peritonitis supervene in enteric fever in which no such change occurs, and when the symptoms and general state of the patient might obscure the evidences of so serious a complication. In estimating the degree of intestinal paresis, the late Mr. Greig Smith proposed a method to which some have attached great importance. This was carefully to auscultate the surface of the abdomen for five minutes, and if at the end of that period no sound of gurgling or the like had been

heard, a complete paralysis might be assumed. Of the diagnostic importance of indicanuria, Nothnagel thus expresses himself:—"I consider the excretion of large quantities of indican to be one of the most constant signs of acute diffuse peritonitis; in fact, I should always hesitate about diagnosing this condition unless there were greatly increased quantities of indican in the urine."

The two conditions for which acute peritonitis might be mistaken are intestinal colic and acute intestinal obstruction. Inasmuch as the severe pain of peritonitis is associated almost always with great tenderness over the abdomen, a distinction from *colic* is readily furnished, since in the latter affection the patient is apt to find relief from bending the trunk over the thighs as he sits, or pressing his arms across the belly. It should be remembered, however, that the pain of perforation of the stomach or bowel, which is probably at first in the nature of colic, may sometimes be lessened by the adoption of a like attitude, before, that is to say, the peritoneal inflammation has set in. Moreover, the colic pain is essentially paroxysmal and not continuous as that of the more serious disease, and it is as a rule unaccompanied by vomiting, or by rigidity and immobility of the abdomen, or by marked indicanuria. Pyrexia also is absent excepting very occasionally in cases of lead colic, which superficially resembles peritonitis in the intensity of the pain and in being sometimes accompanied by tenderness. Other forms of colic, hepatic or renal, should offer no difficulty in their discrimination from peritonitis if attention be paid to the locality of the pain and the direction in which it radiates, together with the points above mentioned.

From *acute intestinal obstruction*, the diagnosis of diffuse peritonitis is admittedly not always so easy, and especially since the peritoneal inflammation frequently follows on occlusion of the bowel; but before the two conditions coexist, the colicky rather than continuous character of the pain, the absence of extreme tenderness, fever, or abdominal rigidity, and the early development of meteorism, with vomiting that is copious rather than small in quantity, are all in favour of obstruction; and should there be any visible evidence of increased peristalsis, this would strongly support the same conclusion. Tenderness of the pelvic peritoneum as ascertained by digital examination of the rectum or vagina has been looked upon as evidence of peritonitis, but it cannot always be elicited even when the inflammation is certainly present. It is in the differentiation of these two morbid states that indicanuria fails to be of any assistance, since it is considerable in both.

Among the conditions determining severe abdominal pain that might be taken for acute peritonitis and that are of less frequent occurrence are acute pancreatitis, the torsion of a floating kidney, the rupture of a gravid Fallopian tube, or the twisting of an ovarian pedicle. All of these are characterised by a sudden or fairly rapid onset and great severity of symptoms, among which severe or even agonising pain, more or less continuous in character though with paroxysmal exacerbations, and constant vomiting leading to a state of extreme prostration and collapse, thus

closely resembling acute peritonitis. Indeed, so far as the manifestations referable to the abdomen as well as the pulse and temperature are concerned, the pancreatic affection is practically indistinguishable from the peritoneal disease, and the other maladies mentioned approximate closely to the same. There is, however, in all these, a greater liability for the pain complained of to be localised or at least to be of distinctly greater intensity in certain situations, epigastrium, loin, or lower part of the belly, and still more significant are the evidences of intoxication, which, sooner or later and in greater or less intensity, the peritonitis is sure to exhibit, and that are noticeably absent from the other conditions.

Certain diseases within the chest may be mistaken for acute peritonitis, and even laparotomy has been performed for perforation of the appendix or of a gastric ulcer, whilst the real malady was an acute pleurisy or pneumonia. The acute onset, the rise of temperature, the severe distress of the patient and pain referred to the epigastrium or right iliac fossa, have been the justification for the error, which should be prevented by careful attention to the character of the pulse and the pulse-respiration ratio, together with an examination of the thorax (*vide* p. 1002).

In the differential diagnosis of acute peritonitis no less than in the more difficult task of ascertaining its cause, the history of the case is necessarily important, particularly in respect to the previous occurrence of symptoms which might suggest the existence of lesions such as gastro-intestinal ulceration or appendicitis likely to determine the peritoneal disease.

Some opinion may be formed as to the particular variety of infection that is present in any given case. Thus, the greater prevalence of the pneumococcus in the peritonitis of children and the probability of its responsibility for the abdominal affection where a pneumonia or empyema is also present; the great likelihood of the *Streptococcus pyogenes* being the cause of a puerperal or a fulminant perforative case, though the *Bacillus coli* must not be forgotten in connexion with this last. Some assistance in this direction might be obtained from the appearance of the exudation.

The **Prognosis** of acute general peritonitis is always of the gravest. Even with a more rational treatment based on a more accurate knowledge of its causation and pathology, it still remains one of the most fatal diseases.

Excluding the favourable influence of operative interference to be subsequently considered, it may be said that no case of diffuse peritonitis due to perforation can be expected to recover spontaneously. It is true that a very few examples of this condition have been said to have done so, and this result has been attributed to the stomach being empty at the time of the perforation and the consequent absence of invasion of the peritoneum by toxic material; moreover recovery has even been reported when a typhoid ulcer has led to communication of the intestine with the peritoneal sac, but such fortunate events as these are not to be

counted on, and death may be looked for within thirty-six hours, less often within forty-eight.[1]

No doubt by far the larger proportion of cases of puerperal peritonitis die within a week or ten days, as also do those in which the diffuse inflammation follows an acute intestinal obstruction, appendicitis, or enteric fever without perforation. But a few cases recover without operation, and even where the symptoms have been of considerable severity they may gradually abate and convalescence be entered on after a fortnight or three weeks. Sometimes patients, whose condition would be included in either of these categories, may pass on into a more chronic state lasting for weeks or even months with uncertain issue up to the last. The particular variety denominated progressive fibrino-purulent peritonitis is of this character, and it is to be noted that the exudation in all these forms is purulent, and gradually spreading as fresh areas of peritoneal inflammation are successively developed, fresh outbursts of symptoms occur to subside and recur again. Worn out by repeated hectic attacks, the patient becomes emaciated and presents all the characters of a subacute or chronic septicaemia, which may terminate in death, with or without other manifestions such as pericarditis, endocarditis, pleurisy. Yet even in such unfavourable circumstances as these a case now and then does manage to recover, though with such grave impairment of health as to determine a long period of invalidism.

It will be seen from this account that although a purulent exudation as a rule betokens an unfavourable termination, yet it by no means necessarily does so. Even less hopeful is the peritonitis associated with a serous or sanious effusion, or that in which no fibrinous exudate coats the surface of the serous membrane to arrest or to diminish the toxic absorption, or when a too energetic procedure at operation has widely damaged the protective endothelium or washed away the beneficial white staphylococcus. Reference has already been made to the relative virulence of some of the recognised infective organisms (*vide* p. 916).

With regard to the prognostic value of the individual symptoms, it has to be remembered that death or recovery chiefly depends on the severity of the toxaemia, and it is the indications of this condition that furnish the most reliable data for framing a forecast of the issue. Moreover, it is this underlying septicaemic state which, being common to all forms of acute peritonitis however they may arise, gives to them their similarity, which becomes strikingly apparent in the fatal cases. At the same time, if there be no improvement after the disease has been established for twenty-four hours, even in those symptoms more directly referable to the abdominal lesion, the outlook is exceedingly grave. Thus, if the vomiting be very severe and constant, the condition is proportionately serious, whilst action of the bowels, whether occurring spontaneously and regularly, or easily induced, is to be

[1] Of 70 fatal cases collected by Sir F. Treves, 14 per cent died within 36 hours, 6 per cent between 36 and 48 hours, 20 per cent between the 3rd and 5th day, 33 per cent between the 5th and 7th day, and 27 per cent after a week.

regarded as favourable. Very little dependence can be placed on the temperature for forming a judgment, although a persistently subnormal curve is almost invariably fatal. Nor can the severity of the pain be taken as a guide to the severity of any given case, since it is often most marked in some of those which recover. In proportion also to the rapidity of the pulse and the extent of disparity between it and the temperature, is the gravity of the case. An expression of the extent of intestinal paralysis and of the asthenic condition of the patient, is the degree of meteorism, the serious import of this phenomenon being increased by the mechanical impediment it offers to the action of the heart and lungs and the consequent efficiency of the circulation. By the experienced, much importance is attached to the general aspect of the patient, and although, as previously stated, death may be impending when this would appear to be almost favourable, it is none the less a valuable index of the general condition, of which a deepening prostration and collapse are among the most serious manifestations. It need hardly be added that acute peritonitis of whatever form is most fatal in the very young and the aged.

Treatment.—The rational treatment of acute diffuse peritonitis should be based on the following considerations. First, the recognition of the infectious nature of the disease, and that many of its symptoms and its usually fatal result are the consequences of toxic absorption from the peritoneal cavity; secondly, that within limits the peritoneum possesses certain defensive powers against the organisms by which it may be invaded; thirdly, that the various pathogenetic microbes exhibit different degrees of virulence; and fourthly, that acute peritonitis is almost always secondary, the infection being the consequence of some injury or morbid condition of one or other of the structures which the serous membrane envelops. Notwithstanding that acute peritonitis is of an infectious character and due in some cases to organisms that, developing elsewhere in the body, give rise to maladies of more or less distinctly limited duration, it cannot be said that for purposes of treatment the disease under consideration is to be regarded as one that will recover provided that the patient can be tided over a certain period of time, though it may be admitted that occasionally a case may in its behaviour have suggested a defined course or even an apparent crisis. The toxic effects are too profound and applied over so large a surface as to prevent any such view being taken for practical treatment, which must be immediate, prompt, and thorough if it is to have any chance of success. The indications for treatment that follow from these considerations concern first of all the prophylaxis of the disease, and failing that, the encouragement of the defensive agency together with the removal of the source of the infection as well as of the infective and toxic material that may have accumulated, dealing meanwhile with the symptoms so far as may be possible.

Inasmuch as acute peritonitis, whether general or local, is, possibly with rare exceptions, secondary to lesions of the abdominal organs, it

is obvious that the most efficient measure for the prophylaxis of the peritoneal inflammation would be the prevention of these visceral lesions; but it is equally obvious that our ability in this direction is extremely slight. The prevention of appendicitis, of gastro-intestinal ulceration, or of acute intestinal obstruction is still to seek.

Somewhat more promising, perhaps, is the prevention or arrest of inflammation after the infection has taken place. In connexion therewith, as in respect to other means of treatment, the question of time is a most important one. After the exposure of the peritoneum to the microbic invasion there is an interval—at least of a few hours—before any considerable or even appreciable response on the part of the serous membrane takes place, and it is this period which offers the opportunity for much to be done. It is now that efforts may be made to encourage the natural defensive measures possessed by the peritoneum, to increase the number of phagocytes and the quantity of bactericidal serum. At present it must be admitted that the extent to which this can be accomplished is very limited, but the work of Messrs. Dudgeon and Sargent previously referred to has gone far to shew the way. The beneficent action of their white staphylococcus, which is the earliest to appear after infection has taken place, in promoting leucocytosis should certainly not be arrested by too extensive an irrigation of the peritoneal cavity unless an abundant escape of intestinal contents have occurred. The transfusion of the peritoneal cavity with warm neutral saline solution has been found to stimulate the production of phagocytes, and for this reason has been employed in the early stages of laparotomy. Various substances have been administered previous to operation for the same purpose. Of these nucleic acid has received an extensive trial at the hands of Mikulicz-Radecki, who employed a 2 per cent solution of the neutralised acid, about 50 c.c. of which were injected subcutaneously twelve hours before the operation. Of "45 laparotomies in which the abdominal cavity was exposed to infection by the contents of the stomach or intestines, or by some other infectious secretion, 38 recovered, and in none of the 7 fatal results was peritonitis the cause of death." The impression of this observer, therefore, is favourable to this method of preparation of the patient. It is fair to observe, however, that he attributes considerable benefit to irrigation of the peritoneal cavity with warm saline solution, which he latterly practised.

Next to developing the normal protective functions of the peritoneum would be the combating the infection by means of antitoxic serums. However desirable these means might be, at present they have attained to but a small degree of efficiency. For those varieties of infection characterised for the most part by a moderate virulence, the individual can as a rule produce the requisite antidote, whilst such forms of anti-streptococcic serum as we do possess are useless against the invasion of organisms of this class whose virulence is overwhelming; but for the commonest and most generally lethal variety, the *Bacillus coli*, encouraging results have been obtained by the use of a polyvalent serum, from a

horse immunised against thirty-one strains of *B. coli*, in twenty-five cases of appendicular peritonitis (Makins and Sargent). It is in this direction that a great desideratum exists, and that great benefit may be hoped for. Were it possible to know with which particular organism or organisms the patient was infected, the plan of treatment might more satisfactorily be entered upon, but this cannot be done until the abdomen be opened when the plan of procedure has already been commenced.

Before the real nature of peritonitis was understood or its operative treatment even contemplated as possible, the remedial measures consisted in the withdrawal of food which the vomiting itself almost compelled, hot applications to the abdomen when they could be borne, and the administration of opium. Under the influence of the narcotic the patient's last hours were soothed, and death came painlessly, too often when the apparently improved condition which the drug conferred had given rise to illusive hopes of recovery. Now and again a case, even so far as can be judged coming within the category of what we now term fulminant, escaped the almost inevitable termination, and offered such justification as there was for such a plan of treatment. But with a truer conception of the pathology of the affection other methods of relief were proposed, and the propriety of previous measures was called in question, and surgical interference, rendered possible by Lord Lister, became not only justifiable, but imperatively indicated.

As may well be supposed, a considerable variety of practice has been exhibited by various surgeons in the nature and extent of the operative procedure, and even now there is far from uniformity in this respect. It should be clearly understood at the outset for what purpose operation should be undertaken, and this would appear to be twofold—first, the cutting off of the source and supply of the infective material, for example, by the removal of a gangrenous appendix or coil of intestine, or the repair of a perforated stomach or bowel; and secondly, the removal of the toxic products of the inflammatory process. It will be apparent that the extent to which these indications can be met by operation must vary very considerably in different cases, and the character of the operation requisite for the removal or repair of the source of the infection more properly falls under the consideration of the various lesions responsible.

Here, again, time is a most important consideration. Difficult as the statistics of operation results are to compare, owing to the different conditions present, as well as for other reasons, they all nevertheless agree in shewing that if laparotomy is to be performed, its best chance of success lies in its early carrying out. This is especially true in regard to those cases of acute peritonitis which are caused by the rupture of a hollow viscus, when the effects are sudden and overwhelming. The most favourable time for operation is, as Mr. Symonds insisted, during the "period of repose" which occurs some hours after the initial symptoms.

Before the nature and extent of the defensive mechanism of the peritoneum was at all realised, either in regard to the part played therein by the white staphylococcus, or the protective value of the layer of inflam-

matory lymph, the indications for operation next to the attempted repair of the visceral lesion were taken to be the completest possible removal of the infective material that had escaped into the cavity. When a simple opening of the abdomen was insufficient for this purpose, as was usually the case, more extensive methods were adopted, consisting in free flushing of the peritoneum, and even the withdrawal from the abdomen of the coils of intestines (evisceration), which were cleansed and replaced. The harm done by this handling of the peritoneum by damaging the natural defensive efforts of the membrane, as well as the injury inflicted on the endothelium, not being appreciated, the results of such procedures were calamitous in the extreme; so much so that the mortality almost if not quite equalled that of the cases left alone. Inasmuch as occasionally a case of perforative peritonitis recovered without operation, the justification for such a line of treatment was scarcely apparent. A truer conception of the actual conditions furnished by increased knowledge shewed better results when less severe means were followed, and a reversion took place to the more restricted—often too limited to be efficient—methods that characterised the earliest practice of abdominal surgery, when the peritoneum was regarded as a structure dangerous to interfere with, a relic of the teaching of the pre-antiseptic era. A further objection to the more severe methods as represented by evisceration and extensive irrigation is to be found in the increased shock which they entail, a serious consideration when the initial lesion and the inflamed peritoneum itself are powerfully provocative of such a condition. The principles which may now be taken to furnish the most successful results, deduced from the practice of one possessed of large experience and considerable authority (19), may be thus stated—the removal or remedy of the source of infection, adequate drainage of the peritoneal cavity, the least possible manipulation or exposure of the viscera, and rapidity of operation, every means being taken to reduce shock. For details as to the performance of laparotomy, the site of the opening, the methods to be adopted to ensure adequate drainage, and the after-treatment, reference must be made to works on practical surgery. It may, however, be pointed out that, differing as the results do in the hands of different surgeons, a moderate irrigation of the peritoneal cavity with warm saline fluid appears to be most satisfactory, though, on the other hand, free incision and dry sponging of the peritoneum without irrigation is becoming increasingly advocated. The propriety of irrigation has been thus summed up by a recent writer (C. J. Bond):—"It is beneficial when the peritoneal cavity contains foreign material, such as blood, gastric or duodenal contents, or faecal matter, infected bile or urine, capable of removal by flushing. It is also useful locally for the removal of purulent material at the primary focus of invasion, in which case phagocytes are no longer living and active. It is harmful in the case of fibrinous deposit and sticky coils without fluid, and must be used with the greatest reserve, if at all, in the case of sero-purulent exudates containing active phagocytes." Some operators attach much importance to keeping the patient

in a position in bed which favours the subsidence of the inflammatory products into the pelvis, as far, that is, as possible from the diaphragmatic surface, where absorption of toxins and organisms mainly takes place. This posture is best obtained by raising the patient to a semi-sitting one, with the knees kept flexed by pillows beneath them. In regard to drainage, whilst this cannot be too complete where the more virulent organisms are concerned, the power the peritoneum possesses of dealing with the milder forms should be recollected. It is all-important, for the drainage to be efficient, that the pelvic cavity should be specially attended to in this respect. Age is no bar to operative treatment on these lines, since Mr. Mayo Robson reports a successful result in a man of seventy (19).

Foremost among the measures which are supplementary to operation is the introduction into the system of normal saline fluid. This may be accomplished to the extent of several pints by rectal injections, or, as advocated by Mr. H. Barnard, by the introduction into the subcutaneous tissue of each thigh of a stout brandy-syringe needle connected by india-rubber tubing to a suitable receptacle for the fluid, which should be of the strength of a teaspoonful to a pint (0·6 per cent), the water having been boiled and kept at a temperature of 115° F. By placing the receptacle about a foot above the patient's limbs, the requisite siphon action is obtained, the tubes being previously filled. As much as fifteen pints may be introduced into an adult man in twenty-four hours by this method, eight pints being looked upon as a moderate quantity. By some the fluid is injected into the rectum, by others directly into the veins. It is stated that this method makes the outlook far more favourable in suppurative peritonitis, and that the resulting benefit is due to minimising the shock and its effects, which are closely associated with the diminished quantity of fluid in the vascular system and tissues. The improvement in the appearance of the patient, and in the pulse, which becomes fuller, stronger, and slower, and the relief afforded to the thirst and dryness of mouth are very marked. Moreover, it is affirmed that after considerable transfusion has taken place, the osmotic current in the peritoneum is no longer from the cavity into the vessels, but in the reverse direction, so that the absorption of toxins into the blood is arrested and fluid is poured out into the peritoneal sac. The increased fluidity of the blood in the now fully filled vessels dilutes the toxins that have been absorbed and at the same time promotes their elimination by the kidneys.

Probably no one of the means pursued in the treatment of acute peritonitis has undergone a more complete reversal than the administration of opium. Formerly, when operation was not to be thought of, opium in some form was the routine practice; but when the propriety of opening the abdomen was admitted, the ill effects of the narcotic in masking the symptoms, engendering a false expectation of improvement, and so leading to fatal delay, came to be recognised, and, swinging to the other extreme, the drug was absolutely prohibited, and this is the generally accepted rule at the present time. Some further support for this view is to be found in the alleged diminution of phagocytic activity

after the use of opium or morphine. One result of the use of opium is inhibition of intestinal peristalsis. Whilst, on the one hand, some advantage may be claimed for keeping the bowels quiet in that the toxic materials are less moved towards the absorbing surface of the diaphragm, it is at the same time a question whether the effects of the opium may not have increased the liability to paralytic distension of the gut which is one of the most fatal manifestations of the disease. When, however, operation has been refused by the patient or declined by the surgeon for sufficient reason, it may become necessary to push the administration of opium to its full limits as guided by the state of the pupil. Such a course at least makes the patient more comfortable, and may favour the formation of adhesions by keeping the intestines at rest, and there is always the very remote chance of recovery even among the worst cases. In direct opposition to this constipating line of treatment, which, it may be observed, the withholding of food by the mouth also favoured, was the frankly aperient method introduced by Lawson Tait, and pursued more or less completely since his time. A suggestion for this course undoubtedly exists in the diarrhoea which characterises some forms of acute peritonitis, especially that of puerperal origin. But it is not to be recommended in the earliest stages of the disease, when the source of infection remains unremoved and active movements of the bowels increasingly distribute the infective material throughout the peritoneal cavity, and tend to make general what otherwise might have remained a localised peritonitis; whilst it is expressly contraindicated when acute obstruction or paralytic distension is present. Calomel is for various reasons the most suitable purgative to employ, but among those well qualified to speak great difference exists as to the extent to which it should be given. Thus, Mr. Mayo Robson writes (19):—"After removal or repair of the cause of the peritonitis, one-eighth of a grain of calomel given every hour or two by the mouth, until a grain has been administered, not only helps the passage onwards of flatus, but by its antiseptic effects tends to prevent decomposition in the gastro-intestinal contents and so to arrest the formation of gas." In contrast to this, Mr. Barnard states that the practice followed at the London Hospital for general peritonitis "is to administer a grain of calomel every hour until the bowels are open, and then to maintain a moderate diarrhoea. In some cases as much as 40 and 50 grains of calomel have been administered, and in one case 72 grains were given." Saline aperients, as originally recommended by Lawson Tait, may usefully supplement the calomel when in moderate doses. There is much, no doubt, to recommend this eliminative treatment when judiciously employed, since the contents of the intestine in the circumstances of the peritoneal inflammation and bowel obstruction come to acquire an unduly toxic character.

For what may be said concerning the treatment of special symptoms a few words will suffice, since their alleviation is in great part to be looked for as a result of the general therapy that has been described. The condition of a patient, the subject of acute diffuse peritonitis, is one that certainly calls for rest, which the prostration and pain alone are sufficient

to impose. The most suitable position to be adopted has already been pointed out. Now that opium and its derivatives have been declared inadmissible, the relief of the excruciating pain is a matter of the greatest difficulty in the early stages, but after operation and the real nature of the case is clear, resort may be had to very small doses of morphine subcutaneously, sufficient to allay the severity of the suffering without altogether obscuring the condition. It is not often that the extreme tenderness permits hot applications to the abdomen, but when these are possible they are of much benefit.

The most effective means of maintaining the power of the heart and counteracting shock is the hypodermic injection of three to five minims of liquor strychninae. Marked improvement in the general condition frequently follows this proceeding.

Vomiting may be controlled or at least diminished by a simple effervescing mixture containing three or four minims of dilute hydrocyanic acid, or drop doses of tincture of iodine in a teaspoonful of water. Lavage has been recommended for severe vomiting. But it must be confessed such a proceeding is one of great difficulty in the subject of acute peritonitis, and not only from the distress and discomfort it occasions for the first few times of doing. It should not be forgotten that the vomiting may be due to opium if that drug have been given. The dry unpleasant state of the mouth may be much improved by rinsing with a dilute (1 in 80) solution of carbolic acid in rose-water or listerine, together with frequent applications of glycerin and borax.

The feeding of the patient is ever a difficulty. The frequent vomiting too often prevents anything being given by the mouth, though an occasional teaspoonful of meat essence or jelly, or of milk, may be successfully given by a clever nurse. Should diarrhoea occur either as the result of calomel or without aperient, rectal feeding becomes impossible. It is worse than useless to make the patient swallow ever so little when the stomach contains any of the intestinal contents that have regurgitated into that organ, and it is better then to promote vomiting by full draughts of warm water. It is sometimes possible, when the administration of the saline fluid is effected by rectal injections, to include in the fluid liquid meat preparations partially digested, but this is not efficacious if the bowels are being kept freely open. Since the vomiting is often slight when the bowels are loose, feeding per oram is often possible when diarrhoea is present, but this by exciting peristalsis is not without harm. Subcutaneous injections of sterilised olive oil, or solutions of glucose, or of serum may be given when feeding in other ways is impossible. But abstention from food for the first twenty-four or even thirty-six hours may be borne without much harm being done, and by that time in the worst cases of perforative peritonitis the course of the case has probably become manifest. Afterwards large nutrient enemas, containing peptonised milk, somatose, glucose, and brandy may be given if only a very moderate action of the bowels is maintained. The distressing thirst is very greatly alleviated by the saline transfusion, and this does away with the constant sucking

of lumps of ice, an objectionable proceeding when carried to any extent as exciting the vomiting and adding to the depression by the collection in the stomach of the cold water. An occasional fragment may, of course, be permitted.

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CHRONIC PERITONITIS

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Pathology and Etiology.—The morbid conditions to which the term chronic peritonitis may fitly be applied are conveniently arranged in two groups: (A) Cases following on an acute peritonitis. (B) Cases which, from the beginning, have run a chronic course, occasionally perhaps manifesting acute or subacute outbursts.

A. As with most other diseases which pass from an acute into a chronic condition, no very definite point can be taken from which to date the latter characters. But when on the subsidence of an acute attack of peritonitis the temperature falls, and pain, tenderness, vomiting, and other characteristic symptoms abate, yet complete recovery does not take place, and other, and in the main, less definite symptoms supervene, then the case is said to become chronic. In an acute case which has completed its course, and ended in a total absence of symptoms and in restoration to health, the products of the inflammatory process either become broken down and removed by absorption or, as often happens,

membranes and fibrous adhesions which exhibit no tendency to spread are formed as the direct results of the acute condition. On examining the abdomen in later years these adhesions are often wrongly regarded as evidences of chronic peritonitis, particularly since, as will presently be seen, similar structures do no doubt form an important part of the changes brought about by the chronic affection. Certain forms of acute peritonitis, more especially those determined by conditions other than perforation of the hollow viscera, and that are attended with suppuration, may run a course the duration of which may justify their being designated as chronic. Starting in a strictly localised manner, the inflammatory products are at first limited by the formation of adhesions, subsequently extending in an insidious fashion as fresh areas of suppuration are established and fresh adhesions are formed. In this way the greater part of the peritoneum may become invaded in the course of what may amount to many months, neither the severity of the local changes nor of the toxic manifestations being sufficient to cause death, though the general health of the patient steadily deteriorates. Such cases have been previously referred to as "acute progressive fibrino-purulent peritonitis." It is only in the sense of their duration that they are to be regarded as chronic, being rather of the nature of a succession of acute or subacute attacks of localised character. We must avoid any confusion between the static results of past peritonitis and the slowly progressive changes of the chronic disease.

B. The cases which are unattended at their outset by acute symptoms, although these may subsequently arise in the slow progress of the disease, owe their causation to a number of conditions, some of which are most obscure. By far the greater number of cases included within this category are those forms of chronic peritonitis associated with (i.) tuberculosis, or with (ii.) carcinoma, which will be considered subsequently.

(iii.) In a third group the presence of infection is more or less certain, though it may be that the organisms concerned are possessed of but a low degree of virulence, insufficient to produce the severe toxic or even lethal effects characteristic of the acute inflammation, or to determine the formation of those products which anatomically are met with in the acute condition. Many of the cases here included are met with among females, in whom pelvic peritonitis of a slow and insidious nature would seem to be especially common, judging by the frequency with which the structural changes of the disease are seen after death. For some of these no doubt the gonococcus is responsible, an organism that has previously been shewn to be generally milder in its effects than are some other microbes, but the specific germ giving rise to other cases is not apparent. The opportunity for infection of the serous cavity via the female genital passages is obvious, and it is possible that the liability is increased during the menstrual period.

To syphilis must be credited a certain number of cases. Among the various structural changes determined by the syphilitic virus is a widespread fibrous hyperplasia; and as a part of this process the peritoneum may be extensively affected, especially in infants and

children the subjects of the congenital disease. Prenatal peritonitis of a chronic character is probably always due to syphilis; but it is usual to find certain visceral changes, such as cirrhosis, gumma, or even lardaceous disease, coexisting with the peritoneal lesion.

In the remaining groups of cases viewed causally, whilst infection cannot be demonstrated, it most certainly cannot be excluded, and the significance therefore of the various antecedent circumstances which are to be recognised becomes proportionately diminished from a true causal point of view. The effect upon the abdominal viscera, more especially the gastro-intestinal canal, whether of poisons or of injury, always admits the probability of such an impairment of their resisting power as to permit of microbic invasion of the peritoneal cavity and to render what at first sight appears to be a simple chemical or mechanical cause, really only the precursor of a true infection, always admitting that the damage to the peritoneum inflicted by the former agents contributes to the results established by the latter.

(iv.) *Toxic.*—Bright's disease, alcohol, and lead have each been described, in virtue of their toxic character, as causes of inflammation of the peritoneum, and it has further been affirmed that they produce the effect by way of a sclerosis of the arterioles of the peritoneum. Little ground exists for some of these statements. In a small percentage of cases of Bright's disease the post-mortem appearances of a chronic change in the peritoneal structures, both local and general, are to be found (3), and a granular nephritis is looked upon by Dr. Hale White as an important factor in causing that form of chronic peritonitis of which a universal perihepatitis is a marked feature. [*Vide* art. "Perihepatitis," Vol. IV. Part I.]

It is very difficult to assign to alcohol its precise share in the production of chronic peritonitis, since its effect on the serous membrane alone—if any there be—cannot well be ascertained. Like lead and other metallic irritants, like phosphorus and even the mineral acids, alcohol behaves as a "tissue poison" in proportion to the degree of concentration in which it is given (7). Prominent among the morbid changes induced by these agents are the structural changes of a chronic inflammatory character in the smaller arteries, and following these may come a hyperplasia of the connective tissue of the parts to which they are distributed. Such changes, as we shall see, are among those which characterise a chronic peritonitis; and to this extent alcohol may be regarded as capable of exciting such a condition. But the serous membrane does not experience the immediate effects of the ingested poison as do the mucous membranes of the alimentary canal and the liver; and it does not seem to be as vulnerable as the nervous and muscular tissues which are so prone to suffer from the intoxicant. In the exceptional cases, therefore, in which a chronic peritonitis is found associated with alcoholism, it is practically impossible to say to what extent the state is primary and independent of the associated lesions in the viscera, especially in the liver and kidneys, which of themselves are among the causative conditions of

the peritoneal change. As a factor in the production of that form of chronic peritonitis of which a universal chronic perihepatitis is but a part, Dr. Hale White has shewn alcohol to be most important.

No confirmation of the statement that lead is a cause of peritonitis appears, after careful inquiry among those who have had large experience of the results of this poison, to be forthcoming; and I can find no cases recorded.

(v.) *Traumatic.*—Among the injuries which may give rise to a chronic peritonitis, repeated paracentesis is well recognised. Frequently restricted in its effect to the immediate neighbourhood of the puncture, it sometimes determines a general affection, possibly from a lack of strict antiseptic precautions. Post-operative adhesions belong to the same group. In very rare cases the symptoms date from an injury, such as a kick or a severe squeeze, which may have happened sometime previously. Henoch records such a case in a child which proved fatal. How far a lesion of some viscus, rather than a direct injury to the peritoneum, may have been the actual starting-point of the inflammation will generally remain doubtful, and may not be cleared up by post-mortem examination. Long-continued pressure, as from tight lacing, would seem capable of inducing the features of a slow peritonitis, accompanied, it may be, by few symptoms during life. It is very common to find, after death, a varying extent of membranous and fibrous adhesions, more particularly in the neighbourhood of the caecum and ascending colon and of the hepatic and splenic flexures of the large intestine, and it has become the custom to attribute these in great measure to chronic constipation associated with the constant irritation of scybala or of long-continued distension of the bowel. It may be observed that, whatsoever the nature of the injury, the effects on the peritoneum are more likely to be local and circumscribed than general and diffuse.

(vi.) *Extension from Subjacent Structures.*—The starting-point of a chronic peritonitis is not infrequently some morbid change in the parietes or other structures covered by the serous membrane. The visceral changes may be the result of injury already considered, or may be of an inflammatory or ulcerative character. The effects are far more commonly local than general; and most cases of circumscribed chronic peritonitis owe their origin to some primary visceral lesion.

Certain regions of the abdomen are more likely to be affected than others. Of all these the pelvic peritoneum is the most frequently affected, and here the direct dependence of the inflammation upon the condition of the organs is most evident. Next in order of frequency are the hypochondria and epigastrium, where extensive changes of the diaphragmatic peritoneum may be traced to a pleurisy or pericarditis; a consequence to be explained by the close proximity of these structures, and by the direct lymphatic connexion between the several serous cavities. Passing to the under surface of the liver, the peritonitis may be due to a chronic catarrh or to an ulceration (not necessarily with perforation) of the stomach, duodenum, gall-bladder or main bile-ducts, and new growths in

this situation are a frequent cause of the same. Similar lesions of the caecum and appendix, sigmoid and other flexures of the colon, and more rarely of other parts of the intestinal tract, as well as old hernias, may be the determining cause in their respective localities.

The irritation of a prolonged peritoneal congestion with ascites, as from chronic heart disease or from cirrhosis, has been regarded as the cause of local thickenings of the peritoneum of an inflammatory character. Rokitansky and Ziegler both described this condition. But no widespread peritoneal change has been ascribed to such an origin. It is very doubtful, however, whether this be the true explanation, which probably is to be sought in morbid conditions of the viscera themselves.

In this connexion may be mentioned Gersuny's suggestion (quoted by Nothnagel), that the fibrinous remains of the blood effused on the surface of the ovary at the periodic rupture of a Graafian follicle may escape absorption and become organised into connective-tissue bands and membranes. Prof. Welch has also observed "richly vascularised thin layers of new connective tissue, particularly on the pelvic peritoneum, associated with small haemorrhages and blood pigment." He looks upon the condition as analogous to chronic haemorrhagic pachymeningitis.

(vii.) Lastly, there exists a series of cases, on the whole rare but sufficiently well authenticated, the determining causes of which are even less certain than are those of the several groups already considered. For this reason they have been termed "idiopathic," a term which, in reference to certain forms of acute peritonitis, has been already criticised. Louis surmounted the difficulty by setting down all cases of chronic peritonitis which were not due to cancer, nor the direct sequence of the acute condition, to tuberculosis. Apart from the different signification that this term bears now as compared with that which it possessed half a century ago, most observers consider that tuberculosis does not account for all the cases known now by various names, of which "chronic exudative peritonitis," "chronic exudative and adhesive peritonitis," "chronic hyperplastic peritonitis," "chronic indurative and adhesive peritonitis," are but some. For although it is true that chronic tuberculosis may and does give rise to structural changes in the peritoneum closely if not entirely resembling those met with in the conditions under consideration, there is an absence of the characteristic bacillus both in the new-formed tissue and elsewhere in the body as well as of the caseous material so commonly the result of tuberculosis.

It is in Germany, and more recently in America, that attention has been particularly directed to these cases of the chronic indurative or sclerotic type. Virchow, in 1853, seems to have been the first to describe the affection; Riedel has more recently investigated the condition and reported cases; and it has been fully considered in von Bergmann's *Handbook of Practical Surgery*. Among the most important American writers on the subject are Nicholls, Scott, A. O. J. Kelly, and Wetherill, who have given full accounts of cases. It is an essential feature of the disease that it is progressive, and this has been ascertained

by successive operations on the same patient, in more than one instance necessitated by recurrence and extension of the symptoms which were relieved by the first surgical treatment. In this way considerable areas of the serous membrane come to exhibit the characteristic change, and all degrees from a merely localised lesion to a widespread affection are to be found. In one group of cases of this nature, the peritoneal affection is but a part of a change in which other serous membranes, pleura and pericardium, are concerned. Various names have been given to this variety, such as "polyserositis" by Kelly, "multiple progressive hyaloserositis" by Nicholls, and "polyorromenitis" as proposed by certain Italian writers. It is difficult to avoid the conclusion that these essentially progressive changes of a sclerotic character in the peritoneum, whether it be the sole membrane affected or is so in common with others, are of infective origin, though the virulence be of an attenuated character. Dr. Hale White, regarding the condition from the aspect of universal perihepatitis which he looks upon as a part of a chronic peritonitis, lays especial stress on the frequent presence of granular kidney as the most important etiological factor of the peritoneal inflammation. But this does not exclude the possibility that infection is the immediate cause, favoured as it may be by the general malnutrition determined by the renal disease.

The Influence of Sex and Age.—From the great frequency of pelvic peritonitis, a greater number of the subjects of a chronic non-tuberculous peritonitis from all causes are females; and, though no age is exempt, it is certainly more frequent in the first half of life. The evidence of the malady among new-born infants has been referred to, and the existence of an acute idiopathic affection in childhood has been described.

Morbid Anatomy.—The post-mortem appearances in cases of chronic peritonitis exhibit considerable diversity, both in extent and in character. The entire peritoneum may be affected, or the changes may be strictly limited to one or more regions, the rest of the membrane presenting a normal appearance. Although there is this very obvious distinction between general and local peritonitis,—a distinction of considerable symptomatic importance,—the anatomical changes are for the most part identical in the two states, and differ only in extent and degree. In a marked case of general peritonitis no part of the structure may be exempt from the inflammatory changes; and, similarly, when the lesions are circumscribed there is scarcely any part which may not be the seat of them. Without doubt, however, the chronic manifestations of localised peritonitis—other than pelvic—are more commonly restricted to the neighbourhood of the liver, of the spleen, of the caecum and appendix, and of the pylorus; in these situations, also, the affection gives the most marked evidences of its existence, and hence is most interesting clinically. But a peritonitis circumscribed at first may not always remain so; from it the inflammatory process may slowly extend until the whole of the serous membrane may become affected; or its progress may be marked

by acute attacks in which, if death occur, both acute and chronic changes will be seen side by side.

A description of the post-mortem appearances met with in the various forms of chronic peritonitis falls most conveniently under the following heads:—(*a*) The Peritoneum itself; (*b*) the Adhesions; (*c*) Capsulitis; (*d*) the Effusion.

(*a*) *Peritoneum.*—The essential changes in the serous membrane itself consist in varying degrees of thickening due to the formation of newly developed fibrous tissue, which is characterised by a great tendency to contract. In extreme cases the thickening may reach as much as a quarter of an inch in depth, in the form of nodules or continuous layers over the viscera, the mesenteries, and the parietes, of a pearly white colour and cartilaginous hardness, consisting of strata of fibrous tissue that has undergone hyaline degeneration. From this cause the great omentum is frequently much increased in bulk, and, unless fixed by adhesions to the lower part of the cavity, is retracted towards the upper part of the abdomen, rather oftener towards the left side, forming a cord-like structure which occasionally appears to be subdivided into several irregular masses. The mesenteries are shortened, whereby the intestines are thrown into sharp curves and loops, and dragged backwards towards the spine, giving rise to the so-called "peritonitis deformans." The membrane itself loses its normal shiny, transparent appearance, and is dull and opaque; but a mere milky opacity, with no lack of gloss or lustre, is not to be taken as evidence of inflammation, for such a change is apt to ensue after death if the membrane become infiltrated with fluid. On its surface small spots may be seen resembling tubercles to the naked eye. Microscopical examination, however, shews that such is not their nature: nevertheless it is possible that such cases, treated by operation, may have been assumed to be tuberculous. The chronic inflammatory process is also marked by a very distinct pigmentation of the membrane and of the adhesions presently to be described. The colour may vary from a faint grey to absolute black, and is distributed irregularly in streaks and patches over the entire peritoneal surface, parietal and visceral. That chronic venous congestion should increase pigmentation is illustrated under like conditions in the lung, in ulcers of the skin, and otherwise. The changes in the mesenteries and in the peritoneal covering of the intestines produce, in long-standing cases, a remarkable shortening in the length of the bowel, as well as a contraction in its diameter—all of which changes considerably interfere with the normal peristalsis.

(*b*) *Adhesions.*—The inflammatory process, besides producing a thickening of the serous membrane, usually leads to the formation of new material in places where none should exist. Whether as a part of an acute attack, or in the course of the slowly developing chronic changes, the inflammatory products, as regards their solid constituents, primarily consist of (i.) a fibrinous clot, containing leucocytes and perhaps a few red blood-corpuscles, derived from the exudation which is an essential

part of the inflammation. Into this fibrinous meshwork (the leucocytes and blood-cells for the most part perishing and breaking up) grow (ii.) processes of large nucleated cells, the descendants of the proliferating connective-tissue corpuscles of the fibrous framework of the normal peritoneum, and of those endothelial plates which cover the surface of the membrane and have neither disintegrated nor desquamated. The new cell-elements from these two sources permeate the clot, and gradually give rise to fibrous tissue, and so permanent material takes the place of the temporary adhesions which the plastic lymph at first maintained between opposed surfaces of peritoneum within the inflamed area. The "organisation" of the inflammatory product is further effected and established by the growth into these adhering bands of extensions from the blood-vessels of the normal membrane—the adhesions thus becoming constituent tissues of the affected part. The extent to which such adhesions may be found offers the greatest variety; from a few fine threads or membranous films easily broken down on handling, and occurring only here and there, there may be such an extensive formation of cords and bands as to weld the entire abdominal contents together into a mass from which the single viscera cannot be separated. Or this extreme condition may be limited to certain regions: thus the liver and the diaphragm may be so firmly bound together as to be parted only with the knife; or the pyloric region of the stomach may be closely attached to the under surface of the liver and head of the pancreas; or the spleen may be embedded in dense fibrous tissue which binds the organ to stomach, liver, and abdominal wall in an inextricable union; or, again, the caecum and its appendix or the sigmoid flexure may be involved in a mass of similar material. The peritonitis due to syphilis is singularly free from effusion, and the most characteristic and extreme degrees of "peritonitis fibrosa" (non-tuberculous) come within this category; though, on the other hand, the change may be represented by a few bands and adhesions only.

It sometimes happens that the adhesions may be sufficiently complete in one area to restrict the inflammatory process to that region, and thus a general peritonitis may be avoided; to this extent adhesions may be regarded as beneficial in their effects. But far oftener they are a source of trouble and even of grave danger. Tending as they do, after the fashion of scar-tissue generally, to shrink and contract, they may cause kinkings and constrictions of the intestine; or knuckles of the bowel may become nipped under fibrous bands, causing acute obstruction. The more general and extensive the adhesions, however, the less likely is strangulation to occur; nevertheless sometimes, owing to their situation and the traction exerted on them by the structures to which they are attached, the newly formed bonds may yield and stretch. Occasionally they lead to considerable displacement of the abdominal organs.

Although in the majority of cases it would seem that adhesions once formed are permanent, or may even progressively increase, this is not

always so, and tolerably clear evidence exists of their occasional removal by absorption.

(*c*) *Capsulitis.*—Occurring as a part of a general chronic peritonitis are certain special features connected with the liver and spleen. These organs may be covered with a white tough jacket of partially organised lymph, which may easily be peeled off, leaving the subjacent peritoneum intact, if deficient in smoothness and lustre, and the liver substance of normal character. This condition, originally regarded as a special affection of these organs and described by Curschmann as regards the liver by the name of *Zuckergussleber*, or "iced liver," is now known to be but a part of a chronic indurative peritonitis, of which these capsular coatings may be almost the sole indication, or but a part of a more widespread change.

A very similar condition is frequently noticed in connexion with granular contracted kidney, when the thickened capsule is closely fixed by old inflammatory material to the adipose and connective tissue in which it is embedded. It is less certain, however, that this condition is of peritoneal origin.

Spots of thickening due to the friction induced by tight lacing, and resembling those seen in the pericardium, are of common occurrence anywhere on the surface of the liver and spleen; but the morbid change shews no disposition to spread.

The effect upon the underlying viscera of a thickening and contraction of their serous covering is not unimportant. Frequently the primary cause of the peritonitis affects the parenchyma of the organ or organs progressively with the peritoneum; or the inflammation may extend from them to the serous membrane: when, however, the latter is extensively affected the viscera may suffer secondarily, for the most part by the development of an interstitial cirrhosis associated with chronic venous engorgement, and less often by extension from the peritoneum, with atrophy or degeneration of the proper tissue of the organ. A corresponding effect on the lung is known to follow a chronic pleurisy.

(*d*) *The effusion* in chronic peritonitis exhibits the greatest diversity in appearance and in quantity. Sometimes entirely wanting, at other times it may amount to many pints, and indeed the largest accumulations met with occur in this disease. When the viscera are so matted together as to obliterate the peritoneal cavity entirely its complete absence is intelligible, but the quantity when present bears no very close relation to the extent of the adhesions nor to the duration of the disease, whilst it certainly tends to vary in amount from time to time independently of treatment. How far the fluid is free in the abdomen will largely depend on the amount of adhesion existing between the coils of intestine, or between them and the other viscera and parietes: this may be so slight as to offer no resistance to the ready movement of the fluid, or it may confine the effusion to certain regions of the cavity from which it is dislodged with difficulty or not at all. Small collections of fluid may be completely encysted by the completeness of the limiting structures, and they appear less likely to be absorbed than when the liquid is quite free;

the density of the walls of these pseudo-cysts and their very restricted vascularity doubtless explain this retention.

In nature and appearance the exudation offers great variety. Premising that it is essentially an inflammatory product, and not a transudation of a serous fluid which has escaped from the vessels as the result of an increased pressure in the capillaries due to venous obstruction, it exhibits to a greater or less degree the power of coagulability; and, as such, approximates in nature to blood plasma. In some cases this power is but very slight, and only the merest threads of fibrin are seen; in others large flakes and clots are met with in the body after death, or they quickly form in the fluid, which may be drawn off during life. At one extreme we meet with a clear straw-coloured or citron-tinted fluid, at the other extreme with pus; and between them appear all intervening degrees, described by such a term as sero-fibrinous and sero-purulent. Owing to the extreme delicacy of the new-formed vessels which develop in the adhesions they occasionally burst; hence blood in variable quantity may become mixed with the effusion. Cases have also been recorded in which, after repeated tappings, haemorrhage has taken place between the layers of the newly formed inflammatory tissue, more particularly on the abdominal parietes, forming numerous haematomas of varying size. Sometimes the effused fluid may be reddish-brown or of a greenish tint from the presence of biliary pigments. In long-standing cases, or where the effusion is mainly purulent, curdy, cheesy masses are found in the cavity. Some of the cases of that rare form of ascites which is of a chylous appearance, though the turbidity of the fluid is not due to fat, are associated with a chronic peritonitis that is not dependent on tuberculosis or carcinoma. The characteristic feature of the effusion is probably due to the débris of endothelial cells which are thrown off in the course of the inflammatory process.

Signs and Symptoms.—The signs and symptoms of chronic peritonitis, whatever its cause, will in any individual case be largely conditioned by the relative proportion of the effusion and of the adhesions. Hence arises considerable variety in extent of the manifestations, and in many cases such obscurity of evidence that the diagnosis may long be held in doubt, and finally reached by exclusion only. Even when the signs of abdominal mischief are tolerably evident the diagnosis is made rather by a consideration of them, and of the history of the case collectively, than by the presence of any one crucial symptom. Whilst in one set of cases the physical signs may be fairly obvious, in others they may be scarcely appreciable; the symptoms that occur with marked prominence in one patient may be wholly or almost wanting in another; and the exclusion of tuberculosis will always be a difficulty, often a difficulty left unsolved.

The *physical signs* of the malady are usually present to some degree, and are often well marked, though they are apt to vary as the case progresses. The degree of emaciation of the patient largely depends on the duration of the disease, the extent of the digestive disturbance, and

the nature of the inflammatory process—being greatest as a rule when the fluid tends to be purulent. The skin is generally harsh and dry, often it is particularly so over the abdomen. When effusion is but slight or wanting the abdomen is sometimes flattened and retracted; but, as fluid accumulates, it presents all degrees of distension, especially if there be any meteorism, which is frequent enough. The distension, unless very excessive, is rarely quite uniform, or even constant in irregularity; there may be rather more bulging over the gastric area from a retracted thickened omentum, or the most prominent region may be in one of the flanks; moreover, as the flatulence varies or the fluid shifts, the contour of the abdomen will change likewise. Irregularities of shape may be equally noticeable when the belly is retracted; and the movements of the intestines are frequently visible. Probably the most valuable evidence is that obtained by palpation. A general sense of increased and often irregularly distributed resistance is communicated to the hand of the observer; and at places a feeling of very distinct hardness, as of nodular but slightly movable lumps; with this there is often a characteristic and quite peculiar sensation, imperfectly described by the word "doughy"; this is caused by the juxtaposition of distended and semi-distended coils of intestine, and firm, resistant tracts of new-formed inflammatory tissue and small collections of liquid. When the effusion is considerable, and not encysted, fluctuation may be perceived; yet in this latter case the evidence of fluid may be very indistinct. In rare cases a friction fremitus may be detected. On percussion, if the peritoneal cavity be greatly distended with fluid, a uniform dull note is obtained; if not, irregular areas of resonance and dulness are to be recognised which correspond to the stomach, the intestines, and the inflammatory products, both solid and liquid; as the liquid shifts with the movements and manipulation of the bowels the dull regions are constantly changing, and it is common to find a resonant flank and a dull flank presently to change places, though, unless the fluid be large and the adhesions few or none, such alterations may be but little marked on altering the patient's position. In cases in which the ascites is very great, the removal of fluid by tapping is followed by reaccumulation, and the removal and recurrence may be repeated over and over again.

Even less uniform than the physical signs are the *symptoms* complained of by the patient. At first they are vague and uncertain, coming on gradually and insidiously, and seldom in such a manner as to indicate even the seat of the malady. When supervening on an acute attack the history of the case may suggest the real nature of the disease, but otherwise it is likely to remain doubtful for a long time. Ill-defined sensations of a dragging character, or other discomforts, now and then amounting to actual pain and soreness, may be the first symptoms which are noticed. Even as the disease progresses the evidence of pain is most variable; at times and for long periods it may be almost or quite absent, at others distressing and severe; possibly it varies as fresh tracts of the peritoneum become invaded or acute exacerbations of the malady occur. It seems

pretty certain that the inflammation of a previously healthy serous membrane gives rise to more severe pain than when the structure is already in a condition of disease. Pain, when present, is often aggravated by movement, especially by walking downstairs; and relief is often felt by the firm pressure of a bandage or other support. The abdomen is generally tender to the touch, at any rate in places, and at times this may even render contact with the bed-clothes unbearable; but no certainty or regularity can be affirmed of this symptom. The temperature may remain normal for weeks, or even subnormal, now and then interrupted by irregular attacks of pyrexia, when the thermometer may register as high as 103° F. Sooner or later the bodily strength is impaired; and a slowly progressive weakness is notable, with disinclination to exertion. The pulse is small and quick, and respiration is apt to be more frequent than normal from the impediment offered by the state of the abdomen. The greater number of the symptoms, as might be expected, are concerned with the digestive function and the alimentary canal. The appetite as a rule is poor, though sometimes it is scarcely affected; but the taking of food frequently determines attacks of colic, which are due to spasmodic contraction of the bowels, hampered as they are in the rhythm of their peristalsis by the adhesions. In this way collections of flatus, the result of arrested digestion, become locked up in sections of the canal, giving rise to pain and discomfort until released. All degrees of constipation, often amounting to such complete obstruction as to necessitate operative interference, are produced by the same cause; the shortened bowel may be kinked or twisted, the muscular coat atrophied by the pressure of the thickened peritoneal covering, and bound down here and there by fibrous bands; beneath one of these a knuckle of the tube may become acutely constricted, and, indeed, in every way impediment may be offered to the normal action of the intestine. Vomiting and diarrhoea are very occasional in their occurrence.

The presence of bands of fibrous tissues, the adhesions of the viscera to one another and to the parietes, lead to very varied pressure effects; thus, oedema of the lower extremities, thrombosis, or neuralgic pains may result from pressure on the large vessels and nerves in the abdomen; or jaundice may follow from compression of the main bile-duct; or micturition may be rendered difficult and painful, or the adhesions may be responsible for an intractable dysmenorrhoea. To remember that abdominal pains, often localised, and in their manifestation simulating gastralgia, gastric ulcers, hepatic or even renal colic, may depend upon old adhesions, is of great practical importance; especially as such cases are far from uncommon, and not infrequently amenable to treatment. The diagnosis of such adhesions, however, is generally difficult, and often to be arrived at by exclusion only. As the result of considerable observation I have been led to suspect the presence of adhesions in cases in which abdominal pain is the sole or at least most prominent symptom, and that is definitely associated with gastro-intestinal movements, as for instance during the progress of gastric digestion or occurring in connexion with, especially

after, the action of the bowels. The abnormal adherence of viscera to one another and to the wall of the abdomen might not unreasonably be supposed to interfere with the blood-supply of the organs; and in this way uterine adhesions within the pelvis may explain some cases of menorrhagia.

The progress of chronic peritonitis is rarely if ever towards recovery. The symptoms may abate, but the anatomical conditions for the most part remain, to be a constant source of fresh outbursts of subacute or even acute character. When the clinical indications are at all well marked the course of the disease is almost always to an unfavourable termination, though this may not be reached for several months or even for years. The causes of death are usually wasting and exhaustion, aggravated in neglected cases by bed-sores, or by a chronic suppuration discharging possibly into the bowel, or elsewhere.

Diagnosis.—From the clinical features described, their great range of variability, and oftentimes uncertain character, the diagnosis, as I have already said, is frequently difficult; the exclusion of the presence of tuberculosis is doubtful at all times, and often impossible. The evidences of the condition which are probably the most conclusive, are the peculiar appearance and feel of the abdomen. If the peritoneal cavity be fully distended with fluid, even these characteristics may be wanting, and a precise diagnosis of the real nature of the state must necessarily remain in abeyance. A consideration of the causal conditions which I have fully described shews that the history of the case may sometimes assist in the diagnosis, at least so far as to suggest the probability of peritonitis. Even in the most experienced hands mistakes are not unlikely, especially in confounding the lumps caused by the inflammatory thickening with masses of sarcoma or carcinoma: the abdomen has now and then been opened in the full expectation of finding a neoplasm, but only to discover an extreme condition of peritonitis fibrosa.

Prognosis.—No very general statements can be made regarding the prognosis of the cases here described. At the best, intervals of relief may be hoped for but scarcely promised, and complete spontaneous cure, when the symptoms are at all pronounced, is not to be expected. The greater gravity of those cases in which suppuration continues has been already referred to; and in marked contrast to these are the cases—not a few in number—in which the anatomical evidences of the disease, of which scarce any symptoms were manifested during life, are revealed only on post-mortem examination. The injurious mechanical effects of the adhesions and bands are but ill compensated, as a rule; and their results are therefore lasting, and constitute an ever-present risk to life by causing intestinal obstruction: whilst the less acute phenomena, such as pain, chronic suppuration, and digestive failures, tend to serious impairment of the general health. One point in regard to the prognosis of those cases of chronic peritonitis described by Dr. Hale White which are characterised anatomically by perihepatitis, and clinically by the presence of ascites, is the more favourable course they run after tapping than do those in

which the dropsy is due to alcoholic cirrhosis without any peritonitis. The latter patients, according to this author, hardly ever live sufficiently long to permit a second operation, whilst should life be so prolonged as to admit of it "the diagnosis of cirrhosis of the liver is almost certainly either completely wrong, or else the patient has something else the matter with him in addition to cirrhosis; and, further, it is highly probable that he has some form of chronic peritonitis, of which what is ordinarily called perihepatitis is merely a variety."

Treatment.—The main indications are to remove the inflammatory products as far as possible, and to maintain the general health of the patient. The former object can seldom be attained completely. Extensive fluid effusions should be removed by paracentesis; and the operation should be repeated without hesitation as often as necessary. When the exudation is simply serous permanent relief may be looked for after one or two tappings; but such a result rarely follows if the effusion be at all purulent. Efforts may be made to obtain absorption of the products by mercurial or other applications to the abdomen; of these inunction with unguentum or oleatum hydrargyri, or linimentum hydrargyri sprinkled on lint, and painting with tincture of iodine, are the most usually employed. These measures, however, can seldom be used for a long time, as they are liable to blister or to produce the effects of mercurial poisoning; and more benefit is to be obtained from the free application of a dilute solution of tincture of iodine (one in three parts of water) night and morning, an application which, as a rule, can be tolerated for a considerable period.

The pain and sensations of discomfort, when acute, frequently call for treatment; hot fomentations, either sprinkled with tincture of opium or applied to the abdomen which has been thickly smeared with a pigment composed of equal parts of extract of belladonna and glycerin, usually give relief. Small doses of opium or morphia internally may be necessary to subdue the pain, which tends, unless checked, to continue, and thereby to interfere with the sleep, the appetite, and the general well-being of the patient. As a rule pressure is well borne, and much benefit and comfort follow the wearing of a tightly adjusted belt or coil of flannel; the vague but distressing symptoms comprised within the word "sinking" are much relieved thereby, probably by counteracting the dragging exerted by the peritoneal adhesions, or it may be by emptying the mesenteric venous reservoirs. The impunity with which, under proper precautions, the abdominal cavity may be opened, warrants the performance of laparotomy when the pain is persistent and intolerable, chiefly with a view to a separation of adhesions. Even if these have proved too extensive for complete division, very distinct relief has been known to follow the mere opening of the abdomen, whilst the risks of such a procedure are now reduced to a minimum. How far the operation may determine the permanent benefit which, as will be presently seen, frequently follows it in cases of tuberculous peritonitis remains uncertain; we have not records sufficient to determine the matter, but at first sight it is not

likely that such satisfactory results would follow this treatment in non-tuberculous cases as in those in which the affection is primarily peritoneal; the remote causes of the cases now under consideration are scarcely to be influenced by the simple opening of the abdomen.

One of the most troublesome symptoms is the constipation, which may become extreme. Violent purgatives are certainly to be avoided; as a rule the milder vegetable cathartics, such as senna, cascara, and the like, appear to act more satisfactorily than the saline aperients, and from time to time these drugs may be supplemented by enemas of castor oil.

The general health should be sustained, as best it may, by such food as may combine the most nutriment with the least indigestible residue; hence milk, eggs, cream, cereal foods, soups, and small quantities of meat should form the staple diet. Syrup of the phosphate or iodide of iron, and Easton's syrup, are among the tonics which may be given with most advantage, together with maltine and cod-liver oil.

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TUBERCULOSIS OF THE PERITONEUM

(TUBERCULOUS PERITONITIS)

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The specific form of new growth called "tubercle," which is determined by the invasion of the peritoneum by the tubercle bacillus, may and usually does give rise secondarily to an inflammation of the serous membrane—tuberculous peritonitis. The varieties in clinical manifestation and in post-mortem appearances exhibited by the cases comprised under this name are due mainly to the different degrees and

extent of the inflammatory process, and to the proportions in which the various inflammatory products are formed. At the one extreme the affection consists of grey granulations only, or is associated with so little inflammation as to be scarcely obvious; at the other the true tuberculous element is so surrounded and embedded in the structural results of its own changes and of the accompanying inflammation that it can hardly be found. Statistics of the frequency with which the peritoneum is invaded in all cases of tuberculosis vary so much as to be of no practical value.

It will be more convenient to describe the anatomical conditions met with in the disease before discussing the manner in which it originates, and its relation to tuberculosis of other regions. Except in so far as concerns the etiology of this condition, much that has been said in respect to the morbid anatomy and symptoms of chronic peritonitis is applicable likewise to the present form of peritoneal inflammation.

Morbid Anatomy.—The simplest appearance is that presented by *miliary tuberculosis of the peritoneum*. Scattered over the membrane are numerous small pearly translucent nodules, from the finest perceptible up to the size of a pin's head; usually discrete, with here and there several confluent—the well-known "grey granulation," "grey granuloma," or "miliary tubercle." There may be but very few, the entire serosa may be so covered with them as to give a distinctly granular feel to the touch, or they may be limited to either the parietal or visceral layer, and in the latter situation may be restricted to areas corresponding to tuberculous ulcers of the mucous membrane whence they may spread, by direct extension, to the hepatic or splenic coverings, or to the surface of the pelvic viscera; or again, if widespread, they may be most abundant over the diaphragm, the mesenteries, and omentum.

This process being essentially acute or subacute in its course, caseation is rare, and there is either no coincident inflammation, or at the most it is represented by a small zone of hyperaemia surrounding each granulation, with points of haemorrhage, and perhaps a slight dulness of the normal shining surface of the peritoneum with some desquamation of the endothelium and "stickiness" to touch due to the merest film of fibrinous exudation, sufficient at spots to cause the slightest adhesion of adjacent layers. When the tubercles have been of long duration there may be a deposit of pigment around them, together with a delicate translucent material, the earliest stage of the fibroid change. In many cases there is an effusion of straw-coloured or sanguineous fluid, often amounting to many pints, and differing from the serum of simple ascites due to portal obstruction by being more or less coagulable; it often forms a firm jelly after withdrawal. Rarely is the exudation sero-purulent in character. The mesenteric glands are enlarged and the seat of tuberculous invasion.

Such are the appearances when limited to the specific tuberculous elements of the disease, but just as these "grey granulations" are themselves the structural response to the irritant represented by the tubercle bacillus, so in turn the granulations are associated with inflammatory

changes of varying extent in the surrounding membrane. Certain alterations, moreover, occur normally in the tubercles themselves, and contribute to the anatomical appearances which characterise the disease. The most important of the changes is the formation of fibrous tissue; another consists in the caseation of the new growth, and to some extent of the inflammatory products; and, lastly, an effusion of fluid is a common feature of the inflammation. The degrees to which these various morbid manifestations are present constitute the anatomical differences met with in the disease, and mainly determine the clinical varieties. The duration of the malady is obviously an important factor in determining the extent to which these appearances are developed; for while in the acute cases, as already seen, little more than the tubercles are formed, in those which have lasted some time, the neoplasm is combined with the structural alterations, even to the extent of being almost obscured. But besides the formation of new tissue (fibrosis) and the degeneration of the tubercles (caseation), the perverted nutritive processes known as inflammation entail, to a greater or less extent, an actual destruction of substance by molecular necrosis (ulceration); and this condition is frequently met with in chronic tuberculous peritonitis, of which, indeed, it may be a prominent feature. According as fibrosis or ulceration predominate in the progress of the tuberculous peritonitis, may the disease be described as "fibrous" or "ulcerous"; but no marked line of separation exists between these forms which are mutually connected by all intermediate degrees of one or the other condition. In an extreme case of *fibrous tuberculous peritonitis* the individual tubercles are scarcely to be found, embedded as they are in fibrous tissue to the formation of which they themselves contribute by their own conversion into that material as one of the natural directions in the involution of the new growth. Tough, firm, membranous bands and adhesions are thus formed by which the abdominal viscera are matted together and bound to the parietes. The intestinal coils are shortened and contracted, and the mesenteries and parietal layer are considerably thickened and inextricably involved in the general mass. Such a condition is not infrequently the final stage of some cases of miliary tuberculosis of the peritoneum; the continued development of the bacillus being arrested, the fluid effusion is absorbed, and a general fibroid change slowly supervenes. Even the ulcerous form in favourable circumstances may occasionally terminate in this way. Where the extent of adhesion of the organs is not so extreme, the intestines are apt to be drawn backwards towards the spine or towards the right upper region of the abdomen. This displacement depends on the attachment of the mesentery to the lumbar vertebrae being normally directed obliquely downwards towards the right sacro-iliac synchondrosis; hence when the suspending fibres are shortened the intestinal loops are dragged towards the right. The similarity of the condition presented by this extensive fibrosis to that induced by the syphilitic virus is noteworthy.

In the ordinary *ulcerous form* the tubercles exhibit a greater degree of caseation—the other direction in the normal involution of the new growth

—forming collections of cheesy matter up to the size of a marble, together with fibrous bands, and grey granulations recently developed and in all stages of degeneration; and of no less importance are enlarged and caseous mesenteric glands. In this way very considerable masses of solid material are formed which exhibit various degrees of necrosis and breaking down, involving in the ulceration the tissues of the organs with which they are in contact, and which they serve to bind together. Between contiguous coils of intestine, or between them and the abdominal wall, ulcerating areas of this kind may be seen, often establishing communication between adjacent knuckles of bowel; the adhesions forming more or less complete loculi, in which are contained encysted collections of fluid of a sero-fibrinous or sero-purulent character, or even actual abscesses which may be faecal. As in other forms of chronic peritonitis there is a great tendency for the new-formed tissue to become pigmented, greyish or almost black. Occasionally in the caseous masses small calcareous particles are met with. The intestinal walls become softened and easily tear on handling. It is in association with this caseous ulcerating variety that the omentum is likely to form a large irregular cord, being infiltrated in tracts by cheesy tubercle, inflammatory new growth, and even encysted collections of exudation. This omental tumour is usually situated in the upper region of the abdomen along the course of the transverse colon, often more marked towards the left side; but occasionally when the omentum is fixed by adhesions the tumour may be found elsewhere, as in the right iliac and lumbar regions; but the lumps formed as above described may occupy the iliac or hypogastric regions, or extend into the flanks, until the greater part of the cavity appears to be filled by them. These masses may cause oedema of the lower extremities, or even of the trunk, by pressure on the large veins, or by obstruction of their channel by tuberculous invasion of the vessel-walls and consequent thrombosis. In these cases a very characteristic manifestation of the disease is frequently met with; namely, a development of tubercle, with caseation and ulceration, in the connective tissue around the foetal remains connected with the umbilicus, at which a discharge frequently takes place through a sinus leading from the surface to the necrotic area; and as this again may communicate with the bowel as above described, the mischief may produce an umbilical faecal fistula. The integuments around the navel are red and oedematous.

In a small proportion of cases the enlargement and caseation of the mesenteric and retroperitoneal glands may be so considerable as to form large nodular tumours and constitute the predominant manifestation of tuberculosis: the peritoneal affection being quite subsidiary. From the close lymphatic connexion between the peritoneum and the anterior mediastinal glands, those in the first intercostal space may be found to be tuberculous from peritoneal infection.

A rare form of peritoneal tuberculosis closely resembles the condition met with in cattle and known as Perlsucht. Tuberculous tumour-like masses, made up of nodules, lying under the serous surfaces, have been

seen in a few cases, mostly roundish or flattened, but some pedunculated with long fibrous stalks. The nodules, which are aggregated into tumours, vary in size from that of a millet seed to a pea, being more or less necrotic, and are of the consistence of marrow, greyish, and occasionally pigmented in the centre. Some ordinary grey granulations are often seen around the larger nodules, and the peritoneum itself is much thickened with firm adhesions between the parietal and visceral layers. It has been held by some that human perlsucht can only arise from infection with bovine tubercle, and that this particular variety of tuberculosis is a chronic attenuated form, occurring in cattle as the result of a specific attenuating action on the bacilli exerted by them. Cases have been recorded by Bizzozero, MacCallum, Uffenheimer, and others.

An effusion in chronic tuberculous peritonitis is often wanting; indeed, perhaps, it would be more correct to say that this form of peritoneal inflammation is usually "dry." Some amount of exudation, however, is not infrequent, and is generally turbid or blood-stained, or even purulent; when this is so, the pyogenetic infection has gained entrance to the serous cavity through the intestinal walls which are the seat of tuberculous ulceration. In this liability to be associated with pus-formation tubercle differs markedly from syphilis, although in respect to fibroid formation these two chronic infective diseases resemble each other. Tubercle bacilli are not often discoverable in the effusion, even when the tuberculous nature of the disease is undoubted. There is reason to believe that the germs chiefly remain and propagate on the surface of the serous membrane, and are not shed to any great extent into the fluid. The fibrinous exudation may sometimes be considerable, forming a pseudo-membranous layer over the tuberculous area and fixing coils of intestines and parietes together in such a way as to form a cyst of variable capacity containing sero-fibrinous fluid. Such a condition has been observed to extend upwards from the pelvis, giving rise to a fluctuating tumour between the umbilicus and the pubes. More than one cyst of this character may be found in the same case.

The frequent association of chronic tuberculous peritonitis with cirrhosis of the liver has long been noticed; in 121 cases of the hepatic affection examined after death, Dr. Kelynack found tuberculous peritonitis in 12, or nearly 10 per cent. The serous inflammation is secondary in point of time, and is of the fibrous rather than the ulcerous variety; but owing to the portal obstruction produced by the change in the liver there is a coexistent ascites, and E. Wagner has described a curious brownish-yellow colouring of the external surface of the small intestine. The association is far more frequent in men and in alcoholics. Coexistent tuberculosis in the spleen is frequent, and the capsule of the organ which is enlarged is particularly liable to be thickened with tuberculous growth.

A tuberculous peritonitis is very rarely restricted to a small area, though the disease may sometimes be strictly confined to the situation of tuberculous ulceration of the intestines, adhesions binding adjacent parts

together and limiting the distribution of the affection; a similar limitation may be found in connexion with tubercle as it affects the pelvic viscera, more especially the female reproductive organs.

Etiology and Pathology.—Immediately determined as this disease is by a specific organism—the *Bacillus tuberculosis*—we must now inquire by what channels the virus gains access to the peritoneal cavity.

Tuberculosis, it must be remembered, is a local affection which naturally tends to undergo either a fibrous or caseous transformation. In the latter condition it may break down, the bacillus-containing débris being thereby liable to become diffused throughout the body by the vascular or lymph channels, or by direct invasion. By each of these routes may the peritoneum be infected.

The extensive distribution of the grey granulations suggests the blood-current as a course by which in acute miliary tuberculosis the germs may reach the serous cavity, whatever may be the exact way they originally gained entrance to the blood-stream. It is as a part, therefore, of a generalised tuberculosis that the peritoneal affection may be met with, although the membrane is less often implicated than other structures, and frequently escapes even in a widespread invasion.

A primary tuberculous peritonitis is of very rare occurrence—the difficulty of absolutely excluding a possible focus of infection being very great—and the disease is seldom limited to the peritoneum, though it may be confined to the abdomen. Most commonly an eruption of tubercles on the abdominal serous membrane is secondary to pulmonary tuberculosis and caseating lymphatic glands in the thorax, the infection being conveyed, in all probability, by the lymphatic vessels. In a valuable communication on the distribution of tubercle in abdominal tuberculosis, based on sixty cases observed during one year at the Children's Hospital, Great Ormond Street, by Dr. W. S. Colman, nine cases, or 15 per cent, are mentioned as presenting grey granulations limited to the parietal layer of the peritoneum covering the diaphragm and anterior abdominal wall; in only two of these was there any tubercle in the intestines or mesenteric glands, whilst in every one there was advanced caseation of the thoracic lymphatic glands and pulmonary tuberculosis.

In the caseating ulcerous forms of peritonitis there is a much greater frequency of coexisting tuberculous disease of the intestines or mesenteric glands, the thoracic lesions being often wanting. Dr. Colman found seven cases (12 per cent) of this condition, in all of which the mesenteric glands were caseating; in four there was tuberculous ulceration of the intestine, but in one only was there any tuberculosis in the thorax. In such cases the path by which the virus reaches the peritoneum is most likely from the intestines by way of the lymphatic vessels and glands, often without any lesion of the mucous surface; or a direct invasion of the serous cavity, by the rupture into it of a caseating mesenteric or retroperitoneal gland, has been known to occur. Other sites of tuberculous disease from which the peritoneum may become infected are the vesiculae seminales, the epididymis and testis, the

Fallopian tubes, and more rarely the vertebrae or the coxo-femoral joint. Prof. Osler thinks "that in 30 to 40 per cent of the cases in women the tubes are found affected, usually as a primary lesion from which the peritoneum is infected, less often secondary to the peritoneal affection." As a possible but most exceptional mode by which the bacillus may gain entrance to the serous cavity traumatism may be mentioned.

In connexion with the close association of tuberculous peritonitis in its ulcerous form with caseating mesenteric or retroperitoneal glands, it may be well to note the comparative frequency with which these organs are attacked in all cases of the disease. In 127 cases of tuberculosis in children, Prof. Sims Woodhead found these glands implicated in 100 (78·7 per cent), and "in 14 the mesenteric glands only were affected, that is, there was no tuberculosis in any other part of the body. These 14 cases were accompanied by neither ulceration nor cicatrisation of the intestine, and there was no peritonitis"; the gland-lesion was essentially primary, a condition which can probably never be affirmed of the peritoneum. Dr. Colman found tuberculous mesenteric glands in 66 per cent of his cases, and Dr. W. Carr, in a series of 120 cases of tuberculosis in children, noted that the mesenteric glands were affected in 65 (54 per cent); Dr. Carr concludes from post-mortem examination, and having regard not so much to the proportionate frequency of caseation in particular glands as to the group in which the process is most advanced, that is, in which the disease began, "that tuberculous disease starts much more frequently in the thorax than in the abdomen, and certainly far more often in the thoracic than in the mesenteric glands." He also enters a useful caution against "being misled by the far too frequent use of that much-abused term, 'consumption of the bowels,' which, as employed by the laity, indicates simply marasmus due to improper food and very rarely any tuberculous lesion"; on this point Prof. Clifford Allbutt had already spoken in the same sense (9). The frequency with which the mesenteric glands may be found affected after death very considerably exceeds the occasions on which they may be detected as enlarged during life; and Dr. F. Taylor's opinion that "an amount of disease of the mesenteric glands which can be recognised clinically as such—tabes mesenterica—is uncommon as compared with recognisable tuberculous ulceration and recognisable peritonitis" (9), is quite in accordance with general experience. As the chief and primary lesion, the glandular affection apart from peritonitis is clinically rare, whatever be its frequency as a pathological occurrence. Susceptibility of these glands is easily understood when their liability to irritation from even transient intestinal derangement is remembered. Occurring also at a period when their activity is at its maximum, and their liability to disturbance is presumably greatest, circumstances combine to diminish their normal resistance to microbic invasion, and to make them a place of lodgment and growth for such tubercle bacilli as may reach them from the alimentary canal, in many cases apparently without perceptible lesion of the mucous membrane. Dr. Sidney Martin fed animals on tuberculous sputum, and found that

the bacilli passed through the intestinal walls without there being any ulceration (*vide* also art. "Tuberculosis," Vol. II. Pt. I. p. 276). The germs may remain in the glands for a variable time, perhaps without further spread; or some circumstance may favour their dissemination from these glandular foci when the peritoneum, either alone or as a part of a more general infection, may become the seat of tuberculous disease.

When the peritoneal tuberculosis is mainly miliary and specially invades the parietal layer, one or both pleurae may be affected, the disease spreading from one serous membrane to the other. In such cases the tubercle appears to be restricted to these structures and the glands, and not to invade the viscera; this has been explained by supposing that the inflammatory exudation, thrown out on the surface of the membrane, prevents absorption and thereby hinders the spread of the infection by the lymph-channels. The resulting pleurisy is frequently "dry."

The general conclusions to be drawn from the foregoing observations would seem to be that tuberculous disease of the peritoneum is frequent, whether as a part of a generalised tuberculosis or as the predominant manifestation of the disease; that the peritoneum may become infected from the intestinal canal by direct extension from tuberculous ulceration of the mucosa, or, as appears probable, without any recognisable lesion of the intestine, the virus in such cases either passing straightway to the peritoneum, or reaching the mesenteric glands, from which the serous membrane may become subsequently affected; further, that the disease both of the peritoneum and abdominal glands may be secondary to tuberculosis in the thorax, the channel of infection then being by lymphatics or blood-vessels. And a remarkable fact in respect to the association of tuberculous peritonitis with tuberculous disease elsewhere is the comparative rarity with which tuberculous ulceration of the intestine accompanies the peritoneal affection: and, whilst the intestinal lesion as a rule coexists with pulmonary tuberculosis, this latter condition seldom accompanies the caseating and ulcerous form of peritonitis, though it is often found with a miliary tuberculosis of the peritoneum. Finally, by whatever means and from whatever sources the serous membranes of the chest and abdomen may become affected, the disease, though it may spread from one to the other, rarely does so from them to the viscera.

The means by which the organism finds admission to the intestinal canal, whether by food such as milk and meat, or by the swallowing of infected sputum from tuberculous lungs, is discussed elsewhere (see article "Tuberculosis," Vol. II. Pt. I. p. 278), as also the conditions which may diminish the resisting power of the intestinal mucosa and so favour the invasion of the microbe. Among the latter would appear to be intestinal catarrh or chronic engorgement of the portal area, such as would be caused by cirrhosis of the liver; thus the association of the hepatic affection and tuberculous peritonitis already referred to may be explained, or both tuberculosis and cirrhosis may be favoured by alcoholism.

Such of the remoter causes as can be in any degree recognised are not known specially to affect the incidence of the disease on the peritoneum,

but are involved in the general etiology of tuberculosis. Overcrowding and defective ventilation, rickets and the acute specific diseases, especially measles and whooping-cough, are probably the most effective of all such factors. It is very doubtful whether sex affects the occurrence of the disease, although post-mortem records shew a preponderance among males, especially of the fibrous form; and as regards the influence of age, it is certainly very frequent between the ages of three or four and twenty years, when the tissues of the body approximate more nearly to the embryonic type, and may perhaps be presumed to be more responsive to morbid influences; though it is far from being restricted to that period of life, having been met with at an advanced age. On the other hand it is almost unknown under two years of age. Of Dr. Branson's forty cases none occurred in the first year of life; 5 in the second; 14 in the third; 6 in the fourth; the remaining 15 being unevenly distributed among the other years of childhood. Prof. Osler considers it is most common between the ages of twenty and forty, and this view is generally confirmed by authorities. Hereditary predisposition is certainly not well marked.

Course, Signs, and Symptoms.—The cases in which the abdominal serous membrane is found affected with tuberculosis fall into two great clinical groups, the acute and the chronic—a distinction which is for the most part well maintained, notwithstanding that the disease now and then runs a course that would justify its being placed as intermediary between these divisions, and also that many of the chronic cases are liable to manifest acute exacerbations from time to time. The signs and symptoms are, however, mainly due to the peritoneal inflammation rather than to the tuberculosis with which it is associated.

(*a*) *The Acute Form.*—From the foregoing account of the morbid anatomy and pathology of this affection, it is apparent that tuberculosis of the peritoneum, with or without any associated peritonitis, may occur as a part of a generalised tuberculosis; or it may constitute substantially the entire disease. Corresponding in great measure to this difference in anatomical distribution are the symptoms manifested, though not entirely so. Thus, in cases of a general dissemination of tuberculosis it often—probably usually—happens that symptoms specially referable to the abdomen are few or wanting, and only evidences of a general febrile disease are to be recognised. Likewise in many of those cases in which post-mortem examination shews the peritoneum to be the seat of extensive tuberculosis, indications of its presence during life may have been completely absent or were overshadowed by the symptoms due to tuberculosis in other organs.

The clinical features of acute generalised tuberculosis, the frequent absence of signs of local affection, the resemblance in many points to enteric fever, and its lapse into the "typhoid state," have been fully described elsewhere (see Vol. II. Pt. I. p. 297): it remains here to refer to those characters which distinguish the acute disease when the abdominal symptoms predominate. Speaking generally, such symptoms are proportionate to the extent of the associated peritonitis, which has

been already shewn not to be an invariable accompaniment of an eruption of miliary tubercle. By acute cases are meant those which run a course of one or two, rarely three months, and for the most part terminate in death; and among such the sole evidence of implication of the abdomen may be that derived from the presence of an effusion into the peritoneal cavity—the physical signs, that is, of a simple ascites unaccompanied by pain or any other local symptoms. In other cases, often when the effusion is not so great, pain and tenderness over the abdomen, not necessarily in any special region, nor yet constant in position, may constitute a marked feature, and may precede the indications of the dropsy. A flatulent distension of the intestines, amounting perhaps to a condition of tympanites, may be an early symptom, afterwards to be followed by the fluid effusion; this will of itself add largely to the patient's discomfort and intensify the pain, to which also a coexistent dry pleurisy may contribute. Either constipation or diarrhoea may be present, or these states may alternate; vomiting has been occasionally noticed. The general manifestations, such as malaise, headache, pyrexia of a hectic type (though with a very frequent liability for the maximum temperature of the twenty-four hours to occur in the early part of the day), loss of appetite, furred or dry tongue, thirst, hot dry skin, quick small rapid pulse, progressive emaciation, together with signs of tuberculosis elsewhere, are among the symptoms of general tuberculosis; though they vary considerably in the extent to which they may predominate over those which are referable to the abdomen, or be subsidiary to them. As a general rule the onset of symptoms is more or less gradual, though often without any history of previous ill-health; sometimes, however, the commencement is so sudden—possibly from the bursting of a tuberculous lymphatic gland into the peritoneal cavity—as to suggest enteritis or internal strangulation, and cases have been recorded in which an operation for the relief of the obstruction has revealed an unsuspected tuberculous peritonitis (14, 18, 25, 26). Death in these acute cases is rarely if ever attributable to the abdominal lesions, but rather to a progressively deepening "typhoid" condition ending in coma.

(*b*) *The Chronic Form.*—This division includes those cases, far more numerous than the former, which may last for many months; they tend, as a rule, to end in death, but are frequently varied by periods in which the symptoms subside very considerably: on the other hand they may assume an acute character.

The marked differences in the structural changes which distinguish the (i.) ulcerous caseating and (ii.) fibrous forms respectively, determine to a great degree the differences in physical signs and symptoms which are met with in these two classes of cases: between extreme examples of which, however, all intervening grades occur.

(i.) In the chronic caseating and ulcerous form the symptoms come on gradually and insidiously. For a period, perhaps, of several months the patient has been gradually failing in health, with loss of appetite and some wasting, together with an irregular pyrexia—a combination of symptoms which, though suggestive of tuberculosis, does not necess-

arily indicate the abdomen as being specially the seat of the mischief. Soon, however, the patient begins to complain of a feeling of fulness and of vague uneasiness, now and then amounting to actual pain in the abdomen, usually at no special spot, and slight tenderness to touch, sometimes best marked about the umbilicus. The abdomen on examination is found to be somewhat tumid, chiefly from tympanitic distension; but at the same time an irregularly distributed sense of increased resistance is perceived, or it may be that definite evidence of fluid in the peritoneal cavity is forthcoming at a comparatively early stage. As the disease progresses the diffuse pain and tenderness appreciably increase, and an extremely painful sensibility of the skin to the slightest touch is sometimes noticed. The abdomen becomes more and more swollen, in marked contrast to the chest and limbs which emaciate to an extreme degree. The integuments are sometimes dry and harsh, sometimes soft and oedematous, and over the abdomen are not infrequently pigmented—the brownish discoloration being very liable to follow the use of hot fomentations for the relief of the pain. The superficial veins, especially in the lower zone of the abdomen, are apt to be distended and prominent—partly owing to the absorption of the subcutaneous fat which reveals them, and partly to actual hindrance to the return of blood from obstruction in the abdomen. The amount of exudation in this form is seldom very considerable, and what there is shifts with difficulty on altering the position of the patient; it is confined with varying degrees of completeness by adhesions into imperfectly communicating loculi between the coils of intestine or between them and the parietes, giving rise to gurgling sensations on palpation. The physical signs of fluid, therefore, are rarely complete, and its presence is often inferred rather than proved. It is further noticeable that the amount of fluid varies very distinctly from time to time without obvious cause. Sooner or later a characteristic doughy sensation with a sense of fixity of the intestines on manipulation may be detected—sensations described with difficulty although recognised easily; and the swelling of the abdomen becomes perceptibly irregular, due in great measure to the formation of the omental and other tumours above described. It is peculiarly characteristic of these lumps, that they are by no means constant in situation, or at any rate that they are not always equally apparent to the observer on inspection or palpation; the varying degree of distension of the intestines with flatus or faeces, and the irregular and shifting distribution of the fluid in the peritoneal cavity, probably explain this phenomenon. Another sign, and one which when present is strongly suggestive of tuberculous peritonitis, although it does occur in other forms of purulent inflammation of the serous membrane, is a redness and swelling of the integuments around the umbilicus, followed sooner or later by a discharge of purulent fluid which may become faecal in character. Various pressure-effects may result from the enlarged lymphatic glands or the omental tumours, such as pain and oedema in the lower extremities; among exceptional manifestations due to the same cause jaundice has been met with, and even a general bronzing of the

skin, as recorded by Dr. Colman, brought about by the pressure of swollen glands on the left suprarenal without implication of its structure.

Passing on to the general symptoms the temperature is very irregular; often remaining for weeks within the normal range, it may decline to a daily average of 97° F., or even lower; or it may continue for days or weeks at an elevation of one to three degrees. In the greater number of cases gastro-intestinal digestion is very imperfectly performed, as evidenced by the deficient or capricious appetite, the frequent nausea and vomiting, and the marked constipation with occasional outbursts of troublesome diarrhoea, the motions being ill-smelling and of an unhealthy appearance. In children the motions, as often occurs in other wasting diseases, are frequently fatty and free from bile, indications that with the definite existence of peritonitis suggest, according to Nothnagel, a tuberculous origin. The tongue is red and irritable or denuded of epithelium. Not infrequently the patient complains of difficulty or even pain on micturition. The pulse is small, quick, and soft, and the whole condition of the patient is one of increasing weakness and exhaustion, preventing any exertion and confining him mostly to bed. The face is pinched and drawn, and there is a progressive anaemia, to which is attributable the oedema about the ankles so commonly noticed. Leucocytosis is not the rule, and when present it is looked upon as suggestive of a secondary infection. Unless the meninges be invaded the intellect remains clear, but the temper is peevish and irritable. Tuberculosis in other organs will necessarily contribute to the sum of the conditions which the patient may manifest; but the abdominal signs when well marked often overshadow the chest disease even though this be well advanced.

Such are the general features and the usual course of an ordinary case of this class. But the exceptions to this description are many. Thus pain, instead of being a prominent symptom, may be almost or quite absent throughout the whole duration of the disease; it may recur periodically with considerable severity, or at other times may scarcely give rise to complaint: the tenderness is probably more usual in its occurrence. Paroxysms of agonising pain, resembling in character biliary or renal colic, have been known to accompany tuberculous enlargement of mesenteric or retroperitoneal glands (1). The extent of emaciation and general malnutrition will obviously be conditioned largely by the degree to which digestion is disturbed and by the coincidence of pulmonary tuberculosis; in some cases the wasting, at any rate in the earlier stages of the disease, may be less considerable than has been described.

The clinical course of those cases of tuberculous peritonitis which are complicated with a tuberculous pleurisy depend somewhat on which membrane is attacked first. Should it be the pleura there will be the general signs and symptoms indicative of pleuritic inflammation and exudation, usually on one side, though occasionally on both; an interesting case of the latter condition, exceptional in the recovery, is recorded by Prof. Finlay. The chest symptoms may almost completely subside before the evidence of the peritoneal affection appears, or both may coexist:

but however this may be, it is the abdominal state which preponderates and mainly contributes to the discomfort of the patient, though an attack of pleurisy may be the immediate cause of death. When the pleurisy is secondary to the peritonitis the former rarely gives rise to prominent symptoms; and the abdominal pain and tenderness with swelling, as well as the diarrhoea and vomiting and other gastro-intestinal symptoms constitute the main disease.

When cirrhosis of the liver complicates tuberculous peritonitis, the disease, as might be expected, runs a more rapid course. A considerable ascites masks the characteristic abdominal features, the effusion frequently being haemorrhagic. Pain and even tenderness are less marked, the temperature is only sometimes raised above normal, and slight jaundice is present. Pleuritic effusion on one or both sides occurs with tolerable frequency in these cases.

Enlargement of the spleen may be detected, but this sign is likely to be obscured by ascites or by the omental or glandular tumours present. A thickening of the spermatic cord from extension of the tuberculous inflammation may take place in boys with a patent processus vaginalis (Oehler).

As a general rule, cases such as are included within this group run on for a long time, now better now worse; sometimes even for months the patient improves in health, the symptoms abating, and the physical signs becoming less distinctive; then again, without perhaps any apparent cause, the condition becomes more acute in character, a fresh effusion of fluid takes place associated, probably, with a recrudescence of the tubercle, or the gastro-intestinal symptoms increase in severity, and the general nutrition is seriously impaired; meanwhile the caseating and ulcerating processes steadily progress, establishing communications between the intestinal coils, coupled, it may be, with some suppuration, and a hectic temperature or a faecal fistula; all combining to bring about a fatal result from sheer exhaustion and lack of nourishment. In children especially the remissions of symptoms are conspicuous. Now and then, however, cases are met with, even among those in which the signs and symptoms have been tolerably well marked, in which the tuberculous process appears to be checked, the caseating material to be absorbed, and the inflammatory products to be converted into fibrous tissue; thus there is a considerable alteration in the clinical aspect of the case, which then comes to correspond to those to be presently described. Owing to this change which frequently takes place in the anatomical characters of the disease, the post-mortem appearances do not always correspond to the indications of the physical signs during life; in this way a very considerable ascites, of the existence of which no doubt could be entertained, might disappear, and leave very little trace after death.

(ii.) The definitely "fibrous" form of tuberculous peritonitis, whether it has been preceded by the acute miliary stage, or has followed the ulcerous variety just mentioned, or whether, as sometimes happens, it tends towards this form from the first, always runs a very chronic course,

and is marked by symptoms which for the most part depend on the mechanical interference with the abdominal functions by the overgrowth of fibrous tissue which to a greater or less degree binds the intestines and viscera into an inseparable mass. The abdomen is retracted and flattened over the whole or greater part of its area; the costal margin, pubes, and anterior iliac spines prominently project so as to give a hollowed or so-called boat-shaped appearance which is most characteristic; this shape is brought about by the shortened mesenteries and contracted intestines, together with, in extreme cases, some contraction of the solid viscera under the compression of the shrinking fibrous tissue. Pain and tenderness are but slightly marked in such cases, and a persistent and often extreme constipation is the symptom most generally complained of. The impaired function of the gastro-intestinal tract determines a deteriorated nutrition, as shewn by the general wasting of the body, which may be considerable. When the fibrosis is more restricted in area, displacement of organs, or adhesions of the stomach and intestine, or compression of the latter by bands may give rise to the most variable symptoms, among which, however, constipation is almost surely prominent, though the general nutrition may suffer less and emaciation may be wanting.

Diagnosis.—From the foregoing account it might be supposed that tuberculous peritonitis is fairly easy of recognition, but this is oftentimes far from true. Well-marked cases are doubtless diagnosed without much difficulty provided a reliable history be obtainable; but a very large proportion of the cases, whether in child or adult, are not well marked, and the diagnosis may long remain in doubt; whilst now and then in the absence of signs and symptoms during life the existence of the disease is first disclosed at the post-mortem examination.

Excluding those cases which manifest no abdominal symptoms, and in which the features of a generalised tuberculosis are not specially manifested in any one part, and putting aside also those cases in which the symptoms referable to the chest or cranium so predominate over those of the abdomen that these latter can be scarcely said to exist, it remains to distinguish acute abdominal tuberculosis from chronic tuberculous peritonitis of the ulcerous and fibrous varieties.

The similarity of acute tuberculosis of the peritoneum to enteric fever has been already referred to. The malaise and sense of illness, the elevation of temperature and the diarrhoea, often with pea-soup stools, are features possessed by the two maladies in common; and the resemblance is heightened still further by the occasional existence of a little bronchitis and even of headache in the serous affection. The absence of rash, so far as it goes, is in favour of enteric fever; whilst the diazo reaction of the urine may be manifested in both maladies. Probably the most trustworthy distinctions are to be found in the agglutination test, in the limitation of pain and tenderness to the caecal region, and in the temperature curve, in enteric fever. The progressive rise of temperature for the first few days, followed by a persistent uniformity for about a fortnight, a course more or less distinctly characteristic of enteric fever,

is rarely if ever to be seen in acute tuberculosis; in this disease the temperature may be at its maximum within the first few days, or the highest point may not be reached for several weeks; whilst, if the chart for any long period be available, a far greater irregularity will generally be observable in the peritoneal disease. With a careful record for a month, there should rarely be any doubt, but for a fortnight, or even more, the conclusion may be in suspense. Reference has been made to the occasional recurrence in tuberculosis of the maximum daily temperature in the earlier part of the day, and this is seldom the case in enteric fever (see also article "Tuberculosis," Vol. II. Pt. I. p. 299).

Those cases of acute tuberculous peritonitis characterised by suddenness of onset, previous to which symptoms had been almost entirely latent, may closely simulate various forms of acute abdominal disease, the true nature of the malady being only disclosed at operation. "Occasionally," writes Mr. Raymond Johnson, "the disease presents itself with sudden manifestations, resembling those of an intense general peritonitis, and thus it may readily be mistaken for acute appendicitis, which is the only common cause of peritonitis at the age at which the mistake is most likely to be made. In some of the less acute forms of appendicitis the difficulty in diagnosis may be extreme. Again tuberculous peritonitis may unexpectedly be discovered in a case diagnosed as acute intestinal obstruction." Sometimes indeed an actual mechanical obstruction may coexist with the tuberculous disease, at other times the latter is the sole lesion to be found. As an explanation of the onset of acute symptoms in the course of tuberculous peritonitis, Mr. Johnson from his observations suggests that secondary thrombosis in some part of the distribution of the mesenteric vessels may be produced. Notwithstanding, however, the close resemblance that may exist between the tuberculous disease and these other abdominal affections, a resemblance often sufficient to mislead the most experienced, there will commonly be found, when the whole condition is made apparent, that some important symptom has been wanting; thus, the paralysis of the intestines, so characteristic of acute diffuse peritonitis, finds no counterpart in the tuberculous variety, nor is there the copious indicanuria in the latter which is constant in the other disease. The similarity in children of the tuberculous lesion to intussusception usually lacks completeness from the absence of blood and mucus discharged by the bowel. In children also the peritonitis may be mistaken for chronic gastro-enteritis, but the recognition of lumps on palpation of the abdomen should serve to make the diagnosis.

For the recognition of chronic tuberculous peritonitis it is desirable to keep in mind the descriptions of typical forms such as have been set forth. But it is most needful to insist that many cases do not conform thereto. The irregularity of the course of the disease, the frequent intermissions with marked improvement, the exacerbation of symptoms for lengthened periods, the degree of illness of the patient, often out of proportion to the physical signs which may be obscure and far to seek, and the prolonged duration of the malady—indefinite as these characters

may be—are often those alone upon which the diagnosis can be based; and for their appreciation time is requisite.

Certain specific features of the disease may add to the difficulties of diagnosis. Thus, encysted collections of fluid, with induration and matting together of the structures confining it, may simulate ovarian cysts, and nothing short of a laparotomy may settle the question; the same may be said for the omental and glandular tumours which have been frequently mistaken for malignant growths of the peritoneum, or even of the colon or ovary, the disturbances of the bowels or extreme constipation favouring the error. As a rule the position of these masses below the line of resonance furnished by the transverse colon should distinguish them from hepatic enlargements which lie above that line, and their irregular shape should prevent their being mistaken for the spleen, the outline of which, with its characteristic notch, is usually well preserved in the various morbid changes which that organ may undergo. A further distinction based upon the relative distribution of dulness and resonance over the abdomen has been pointed out by Thomayer, and may sometimes be of value if judiciously considered. This is the larger area of resonance perceptible over the right side of the abdomen as compared to the left, due to the greater retraction of the small intestines towards the right determined by the peculiar oblique attachment of the mesentery to the posterior parietes. That it is an absolute sign or always present is clearly not the case, and will not serve to distinguish tuberculous from any other form of chronic peritonitis. Prof. Osler attributes some importance to the frequently prolonged subnormal temperature in many cases of omental tuberculous tumours as a character serving to distinguish them from growths of a malignant nature.

The examination of fluid withdrawn from the abdomen shews a predominance of lymphocytes, and so suggests tuberculosis. The presence of the *B. tuberculosis* would be conclusive, but they are rarely to be found, and their absence does not exclude the existence of tubercle. Inoculation experiments, however, should determine the nature of the disease. Or injections of tuberculin may be employed, and the character of the reaction that follows, or of any variation in the opsonic index that ensues, noted as contributing to the formation of a diagnosis (see article "Tuberculosis," Vol. II. Pt. I. pp. 292-3).

The distinction during life between a tuberculous and a simple peritonitis must often remain uncertain, if the existence of the latter condition be admitted. Now that it is known that a very definite tuberculous affection may subside to a great extent, and leave no indication of tubercle, the diagnosis of "simple" cases will be less frequently made. But so far as physical signs are concerned no distinctive difference is to be expected, since the effusion and inflammatory new growth must give rise to the same objective conditions in both groups of cases, whilst the history and general clinical course too often furnish but indefinite data for a judgment. Valuable information may be derived from the existence of tubercle elsewhere, but, in the frequent cases in which the new

growth is restricted to the abdomen, the difficulty of distinction may be insuperable.

Prognosis.—Tuberculous peritonitis is certainly not the invariably hopeless disease it was once thought to be; and, although it still determines a considerable mortality, the very distinct benefit which follows treatment in a large number of cases has largely diminished its hitherto fatal character. Moreover, the recognition that tuberculosis, in a certain proportion of even well-marked cases, tends to its own cure by undergoing fibroid transformation has rendered the prognosis more hopeful; though it is true that the prolonged life may not be a completely healthy one, on account of the permanent ill-effects brought about by the formation of fibrous tissue within the abdominal cavity, and the oftentimes grave interference with the free movement of the contained viscera. Statistics from various sources would seem to give ground for the belief that spontaneous cure may occur in as many as 50 per cent of the cases. Certainly the majority of these are characterised by the presence of numerous grey granulations with serous or sero-fibrinous effusion, and it is only very rarely that this result can be affirmed of the caseous and ulcerating cases. As compared to tuberculosis in other regions, a larger proportion of cases of the peritoneal affection tend to improvement, whether spontaneously or by treatment, than can as yet be affirmed of cerebral or even of thoracic tuberculosis. In forecasting the course of a given case, the liability to improvement, even for months, with a subsequent recurrence of symptoms and fatal result, must not be forgotten. On the other hand, the existence of pulmonary tuberculosis, pleurisy, cirrhosis of the liver, or tuberculous ulceration of the intestine, would very seriously affect the prognosis. The longer the case has lasted the longer it may last, and improvement may take place even in the most unlikely; a persistent diarrhoea is the symptom most to be feared. In proportion to the extent of the fibroid change in the tubercle and the absence of caseous degeneration, and particularly of an ulcerous process, the prognosis may be regarded as fairly favourable; but the grounds for anticipating the one or the other of these conditions are not yet formulated, except that those cases which are free from fever are the most likely to undergo spontaneous cure.

Treatment.—In tuberculous peritonitis, no less than in tuberculosis elsewhere, much may be hoped for from such hygienic measures as are comprised in the avoidance of infection by food—milk and meat—or air; and as important means of treatment, which should always supplement any other plan adopted, are fresh air, good food, sunshine and seaside residence to which cure alone may frequently be attributed.

What has been said in the foregoing section on the medicinal treatment of chronic peritionitis is equally applicable to the tuberculous, and perhaps with more confidence. Applications to the surface of the abdomen for the relief of pain, and for promoting the absorption of the inflammatory products, and drugs for the attendant constipation and the maintenance of the general health, are as suitable in the one case as in the

other, the indications for treatment being substantially identical. Special benefit in children has been claimed for the administration of creasote, whether by the mouth in milk, or combined with cod-liver oil as a daily enema (30). It seems to be generally held that iodine exerts a destructive action on the tubercle bacillus, and hence the value attached to the preparations of this drug such as applications to the abdomen of the tincture or liniment of iodine. Dr. B. Yeo recommends a mixture of equal parts of iodoform ointment and cod-liver oil to be rubbed into the skin of the belly twice daily, together with the administration three times a day of a pill containing a quarter of a grain of iodoform and half a minim of creasote. The universally admitted benefit from sea air, especially at Ramsgate and Margate, is probably in a measure due to the respiration of an atmosphere charged with iodine. It is supposed that the iodine which is absorbed into the blood is excreted into the peritoneal effusion—as it is into the several secretions of the body—and then comes in contact with the bacillus. The efficacy, however, of all these medicinal methods is difficult to estimate on account of the natural tendency of many cases to self cure; though this fact should not pretermit the fullest trial of acknowledged remedies.

Since 1862, when Sir Spencer Wells opened an abdomen for the removal of a supposed ovarian tumour and found a large quantity of opalescent fluid and a peritoneum studded with tubercles, simple laparotomy has been frequently performed for the relief of tuberculous peritonitis and often with the same satisfactory result, which in that case was cure; and in the great majority of cases very considerable benefit has ensued. A large mass of evidence from all quarters is now available, which is unanimous in establishing the procedure as not only justifiable, but in many cases imperatively called for. The variation in the anatomical characters no less than in the clinical features of the disease has given rise to some difference of opinion, as might reasonably be expected, as to the most suitable cases for operation as well as concerning certain details of the operation itself; but most are agreed in the benefit which follows its performance.

That the expression "cure," as opposed to mere "relief," is in a certain proportion of cases justifiable, follows not only from the fact that numberless instances are recorded in which well-marked and even severe symptoms of tuberculous peritonitis existed and abundant tubercles were disclosed at the operation, and yet recovery from all discomfort and absence of further symptoms for many years resulted, but still more conclusively also from the well-authenticated cases recorded by many surgeons (5, 14, 18, 28), who have found that on a second laparotomy, performed some months later for some other reason, the tubercles, which were but too obvious at the first operation and were proved to be such both microscopically and by inoculation, had entirely disappeared.

Concerning the operation itself, it would certainly seem that a mere tapping of the abdomen by an aspirating needle or small trocar is insufficient; there must be a considerable opening and a free evacuation

of any fluid present. Beyond this nothing can be said to be necessary. Washing out the peritoneal cavity with sterilised water, warm boracic lotion, hot solution of creolin or other disinfectants, dusting with iodoform, or the withdrawal of only a small quantity of fluid and injection of camphorated naphthol into the cavity, have all been practised and warmly advocated; but, as experience has shewn, with no greater advantage than is obtained by a simple opening—free drainage when the fluid is purulent—and closure of the wound without flushing or other manipulation. A slight rise of temperature has been commonly noticed after the laparotomy, lasting some hours or even a day or two: but as a rule recovery from the operation (which has of course been carried out with all antiseptic precautions) is rapid, and improvement in the condition of the patient is soon apparent. Some reaccumulation of fluid requiring withdrawal is occasionally met with. It may sometimes be necessary to remove certain structures, such as the Fallopian tubes or mesenteric glands, which are infiltrated; this apparently can be done without increasing the risk of the operation unless the disease of the glands be very far advanced or the patient's condition be very enfeebled.

Every form in which tuberculous peritonitis occurs has been made the subject of operative treatment, and with benefit. Undoubtedly the most promising cases are those in which there is effusion, whether serous, seropurulent, or purulent—free or encysted; and in such cases laparotomy may almost be regarded as the only proper treatment. Even in the acute cases of peritoneal tuberculosis accompanied by much ascites the procedure may be resorted to, although the result is not altogether so satisfactory as in the more chronic forms. Nor does the presence of tuberculosis in the lungs, unless far advanced, offer a contra-indication (29); temporary benefit at least may be expected. Notwithstanding the risks attendant on opening the abdomen when there is little or no effusion, and merely a condition of widespread fibrous adhesion among the viscera—risks due to wounding the bowel or inflicting damage by tearing away the adhering bands—yet very marked improvement has been recorded as having followed a simple incision through the abdominal wall and closing up the wound. Operation in such cases, however, is scarcely to be undertaken except with a view to relieve intestinal obstruction.

The statistics compiled by different authors fully justify the recourse to operation which with proper precautions is scarcely attended with danger. Thus, König collected the records of 131 cases of laparotomy for tuberculous peritonitis; of these 120 were women and 11 men; 70 per cent being over 20 years of age. In 107 of these the operation was satisfactory, being completely so in 84, or 65 per cent; whilst deaths directly following the operation only amounted to 3 per cent. Lindner collected 205 cases, of which 7·5 were fatal from the operation, the causes being collapse, sepsis, or acute peritonitis. In only 186 of these was the sex recorded, and of them 11·3 per cent were males. Körsch in 1893 found in 358 collected cases, a mortality of 5·59 from the operation, and complete recovery in 250, or about 70 per cent. Less successful results

were obtained by Frees, who only claimed complete success in 5 of 18 cases treated by himself, whilst in 12, though no ill effect was attributable to the operation, no benefit followed. All these patients were females—in some there were complicating conditions connected with the uterus or appendages.

As with the medical treatment, so with the operative, the rule that a very large proportion of the cases tend towards spontaneous cure vitiates somewhat the conclusions drawn from these statistics as to the precise therapeutic value of laparotomy. Indeed, there are not wanting now authorities, notably Birchgrevink, who altogether discard operation, considering that those cases which do improve or are cured as a consequence of it, would equally recover if left alone, and that cases which will not get well of themselves will not do so as the result of operation. Probably the most judicious course to follow is, that whilst every case should be carefully considered with regard to the actual stage of the disease and the extent to which thoracic tuberculosis may exist, operation is to be recommended after medicinal treatment has had full trial, and has not been followed by any benefit.

It may be observed that the great preponderance of females in the statistics dealing with operation in this affection is to be explained by the great frequency with which the Fallopian tubes are the seat of tuberculosis, and by the performance of laparotomy in a very large number of cases to relieve a supposed tumour or cyst connected with the reproductive organs; the nature of the disease only being disclosed by the operation. It is not to be supposed that females are more liable to peritoneal tuberculosis, for if there be a difference the reverse would be a truer statement.

Various are the suppositions to explain the effect of this treatment, but it cannot be said that any are wholly satisfactory. The facts clearly suggest that peritoneal tuberculosis is not possessed of great activity or powers of resistance, and that its natural tendency to obsolescence is readily facilitated by what appear to be but very trifling disturbances of the conditions under which it is developing. Bumm and others have demonstrated that after a simple laparotomy the grey granulations undergo round-celled infiltration and cicatricial changes, with disappearance of the epithelioid and giant-cell elements of the tubercle. Kischenski has also shewn this to be the case by experiments on guinea-pigs. The exact determining factor of this change is the point at issue. The generally credited cause is the removal of the fluid, but it is doubtful if this be the sole cause. With less reason, the admission in the course of the operation of air, or of light (19), or of putrefactive bacteria whereby toxalbumins destructive of the tubercle bacillus are formed (23), have been regarded as the causes; and Duran has recommended, in place of laparotomy, simple paracentesis abdominis, withdrawal of fluid and injection into the peritoneal cavity of dry aseptic air. The *B. tuberculosis*, being aerobic, the asserted bactericidal effect of free admission of air is not easy to explain; nor is it likely that in ordinary operations either

air or light penetrates very far into the abdominal cavity, although direct sunlight is known to attenuate the virulence of the organism of tubercle, or even to destroy it. Irrigation with antiseptic solutions seems a more potent means, but it is by no means essential to the attainment of the desired result.

Quite recently an explanation has been put forward by Prof. A. H. White of Dublin, based on the fact, as shewn by Sir A. Wright, that the opsonic index is lower than normal in tuberculosis, and that it may be raised by the injection of a tubercle vaccin. By laparotomy the fluid effusion which is, relatively to the blood, poor in opsonin, is removed and replaced by a fresh exudation "containing a larger amount of opsonin and other protective substances." As a result of the operation also a rise in the opsonic index is found to occur just as it would from a tuberculin injection, and this is looked upon as "due to the auto-inoculation which takes place in part as a consequence of the disturbance of the infected area, and in part because during the exchange which occurs between the tuberculous area and the blood, bacterial products from the former are probably carried into the general circulation." Owing to the rise of the opsonic index the curative effect of the laparotomy is thus accounted for. Should this be the explanation, it furnishes an additional reason for operation, and further suggests the propriety of raising the opsonic index by inoculation before operation, and its subsequent repetition should the blood examination shew a fall at any time.

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NEW GROWTHS OF THE PERITONEUM

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SINCE the structural elements of the peritoneum mainly belong to the connective tissue series, the majority of new growths will be of this origin. In accordance with this fundamental affinity it comes about that the tumours met with seldom consist wholly of one tissue, whether fibrous, adipose, plain muscular, or sarcomatous (embryonic); they commonly present indications of more than one of these structural elements, and that which preponderates gives the name to the growth. As compared with the other serous sacs—pleurae and pericardium—tumours of the peritoneum and subperitoneal tissue are more frequent; but even here they are far from common.

Originating as they generally do in the subperitoneal tissue, these growths may for the most part be regarded as being situated behind the peritoneum, but it is at least clinically convenient to group them as retro-peritoneal, mesenteric, omental, and parametric, while fully admitting that the connective tissue in these several regions is continuous throughout. But it is obvious that the relations of a growth arising in the perirenal tissue will be very different to those of one that forms in the omentum or mesentery, when it may be properly said to be intra-abdominal. The bearing of these situations upon the propriety of operation must also be apparent.

As regards the proportional frequency of the several structural types, statistics differ somewhat according to the view taken of the nature of those commonly designated carcinomatous. Owing to their mixed character also, there may be a doubt in which group a given tumour should be placed. As giving a general idea, it may be mentioned that of 57 cases of solid mesenteric tumours collected by Harris and Herzog, 16 were set down as carcinomas, 10 lipomas, 7 sarcomas, 2 fibromas, the remaining 22 being distributed among an almost equal number of mixed types. A collection of 78 cases made by Julliard gives fibromas 19, lipomas 13, sarcomas 13, endotheliomas 3, and the remainder of mixed

character. In any considerable series of cases, the majority will be of benign nature. Out of 62 solid mesenteric tumours recorded by the latter authority, 47 occurred in the mesentery of the small intestine; 10 in the ascending mesocolon, 3 in the transverse mesocolon, and 2 in the descending and sigmoid mesocolon.

The greater number of peritoneal new growths are found in females; of 58 cases, only 22 were in males. The majority are met with between the ages of 40 and 60; yet out of 55 cases 7 occurred in children under 10 years of age.

There is considerable variety in the character and extent of the symptoms met with in connexion with these tumours; this depends on their size, situation, and malignancy. Not infrequently they give rise to no discomfort, or at most to attacks of colicky pains. In other cases the suffering may be much more severe and constant from dragging on the peritoneum; or the symptoms may supervene suddenly and with considerable violence, and closely resemble those of acute intestinal obstruction, which condition indeed may actually be induced by a growth of the peritoneum. When very large, they may cause symptoms due to the pressure they exert on the diaphragm, bladder, bowel, or large vessels. Their diagnosis is often extremely difficult, especially those which are retroperitoneal, and it is very seldom that the nature of the growth, even if its precise situation be determined, is other than a matter of inference. Removal by surgical means is their sole treatment, and even this is not to be attempted when they are malignant, and must be very gravely considered when they are large in size, and retroperitoneal in position, whatever may be their structure.

Fibroma.—Disseminated fibroid nodules, varying in size from a millet seed to a split-pea, are occasionally met with over the peritoneum —parietal, mesenteric, and visceral. They may be many in number, or but two or three. Not infrequently they represent tuberculous granulations which have undergone fibrous involution; but some may be primary new growths of fibrous tissue. "The fibromas merge, on the one hand, into the lipomas, on the other into the myomas; and it is probable that many of the older reports of fibrous tumours, more especially the large ones, really refer to myomas. Purely fibrous growths may, however, develop in the subperitoneal tissue, or may have arisen in the first instance from the symphysis pubis, or other parts of the fibrous tissues of the abdominal pelvic wall, and have become detached into the subperitoneal interspaces" (4). Pure fibromas are usually small, and give rise to no symptoms leading to their detection during life. But Mr. Doran (12) successfully removed a tumour springing from the ovarian ligament, weighing upwards of 16 pounds, which proved to be "a soft oedematous fibroma." In a further communication he refers to several other cases in which the growth varied from 3 to 7½ pounds, and was purely fibrous in structure. Occasionally the tumour is fibromyxomatous. A specimen of this nature, weighing 13 pounds, and growing from the mesentery, was taken from a man by Shepherd of Montreal; although

nearly 8 feet of inseparably adherent intestine were removed with the growth, the patient recovered.

Certain growths of identical structure to the foregoing, or resembling spindle-celled sarcomas, which develop from the muscular sheaths, tendons, and aponeuroses of the abdominal wall, may, when situated very close to the pelvis—as they more commonly are—be mistaken for intra-abdominal tumours, and have been operated on under this impression. These parietal (desmoid) tumours have been described by Doran (13), Stavely, and Olshausen among others.

Lipoma. Fibro-lipoma.—Localised overgrowths of adipose tissue—frequently with some relative excess of fibrous tissue—are among the most frequent of peritoneal and retroperitoneal neoplasms. The conditions determining their origin are quite unknown, but they seem to be distinct from those which lead to that diffuse development of fat which we know as general obesity. These growths are far more frequent in women than in men, in the proportion of 25 to 16 (1); with rare exceptions they have been met with in adults only.

The subserous adipose tissue in the neighbourhood of the kidneys and iliac fossae—with a slightly greater frequency on the right side—is the usual situation from which these tumours spring, and the majority of them continue to be retroperitoneal; from this position, however, while retaining more or less completely a connexion with the seat of origin, they may penetrate between the layers of the omentum or mesentery. More rarely definite tumours of this nature originate in the subperitoneal tissue of the mesenteric or omental folds, where a general fatty overgrowth in varying degree is frequently observed. An enormous fibro-lipoma, weighing 55 lbs., developed between the layers of the omentum, and removed after death from a woman aged sixty-three, was described by Cooper Foster; and Mr. Meredith successfully operated on one in a similar position which weighed 15½ lbs. Sir F. Treves (47) removed a lipoma weighing 4½ lbs. from between the layers of the broad ligament, a very exceptional position, and one where adipose tissue is rarely if ever seen, even in extreme obesity. Two similar cases have been met with on post-mortem examination. To be included within this category are the fatty tumours which correspond with greatly enlarged appendices epiploicae. Springing from extremely slender or broadly sessile attachments, they have been met with completely detached and lying free in the serous sac, although it is very doubtful whether the separation occurred long before death.

An exceedingly interesting and, from a surgical point of view, most important group of subperitoneal lipomas are those which extend outside the abdominal cavity at the inguinal or femoral rings or in the linea alba, where they closely simulate omental hernias. They have been known to attain enormous size and a weight of many pounds; and containing, as they frequently do, more or less extensive prolongations of the peritoneum, they may, even when very small, be the cause of pain, vomiting, and other symptoms which are relieved by their removal.

The greater number of cases have been met with between the ages of thirty and fifty: but children or indeed infants are not wholly free. Lebert records the removal of a fatty tumour which occupied the left side of the abdomen, and had grown with the growth of the patient since it was first noticed at the age of six months; and Lauwers records one, removed at the age of seven years, from a child in whom it had been recognised since a fortnight after birth; its weight was 6 lbs., almost a third of the total weight of the child after its removal. Cima has described another in an infant aged twenty-two months.

It is rare to find more than one of these growths, although the co-existence of several is not entirely unknown (24). As the masses are often multilobed, the portions being partially separated, some of the largest of these tumours may have been formed by a fusion of several at first distinct. There is great variation in their size which, so far as the clinical histories shew, is not always proportionate to their recognised duration. Several have been recorded weighing upwards of 55 lbs. Masses of this bulk have appeared to fill the abdominal cavity completely, displacing the intestines to the sides; if, however, the mass have originated behind the peritoneum, either in the perirenal tissues or at the root of the mesentery, some part of the colon, usually the ascending or descending, may be stretched over the front of the tumour. This is not the case when the growth is developed in the omentum or mesentery.

In structure these growths exhibit much difference in the relative proportions of the connective tissue and fat-cells of which they are composed, the former material sometimes being very considerably in excess. As a rule, being less vascular, they are firmer and paler than healthy subcutaneous adipose tissue; the perirenal fat, it may be observed, normally tends somewhat in the same direction.

The majority of tumours of this class, however, exhibit a greater or less proportion of other material; thus, besides the fibroid tissue, mucoid or myxomatous tissue is of frequent occurrence, so that a myxo-lipoma is almost as common as a pure lipoma. Cartilage has very rarely been found in association with a fibro-lipoma. Areas of calcareous degeneration are sometimes seen, and even true bone—osteo-lipoma. It is possible, however, that these last are to be regarded as foetal inclusions in which the other tissues have undergone fatty change, rather than true lipomas in which bone has developed. Sometimes certain parts of the mass present all the microscopic appearances of sarcoma which is both histologically and genetically a term of the connective-tissue series. In a case reported by Adami and Gardner, there were two retroperitoneal growths, one being a myxo-lipoma and the other a chondro-myxo-fibroma. Whilst some exhibit a very considerable degree of capsulation, and are "shelled out" with comparative facility, others blend in their attachments with the subserous adipose tissue from which they spring, and are marked off by no definite limits.

The *physical signs* of these tumours and the *symptoms* to which they give rise are seldom very distinctive, and their nature is rarely diagnosed.

Usually they have been mistaken for ascites, or for ovarian cystic tumours; and paracentesis abdominis has been frequently performed for their relief without result. Nor is the error to be wondered at. When of large size and distending the abdomen from pelvis to thorax, chiefly in an antero-posterior direction rather than laterally, their soft doughy feel with an indistinct sense of fluctuation may easily be confounded with fluid, either encysted or free. If there be resonance in one or both flanks the signs of ovarian cyst are closely simulated, whilst, should the flanks be dull, the condition might be explained by a large collection of free peritoneal fluid. When there is an area of resonance in front of the mass, due to an adherent colon, and dulness reaches into one flank, the growth may with reason be considered as connected with the kidney, from the adipose tissue around which organ it may have developed. The smaller growths are freely movable, for the most part painless, and give rise to no symptoms; and even with the largest masses little beyond a sense of fulness and weight is complained of. Some compression of the mesenteric and other vessels may be induced by the growth, and even ascites subsequent thereto has been noticed; but there is very seldom any peritonitis. As a rule the patients are in fairly good general health and well nourished: but in cases attended with much emaciation a diagnosis of malignant disease has been made, and any attempt at relief by operation refused on this ground. When the tumours have undergone myxomatous change, and still more when the fatty growth is complicated with sarcoma, the masses tend to grow with considerable rapidity and to interfere the more with the general nutrition of the patient: so that a diagnosis of malignancy is increasingly probable (37). The surface of the mass is usually smooth, though sometimes it is deeply lobulated: it is but little tender on handling, if at all. When of considerable size, symptoms due to pressure on the intestinal canal or on the bladder, or to interference with the free action of the diaphragm, may arise and attain to a severe degree; more often oedema of the lower limbs occurs, sometimes more marked on the side from which the tumour sprung.

The data on which a *diagnosis* may be made are to be gathered from the foregoing description of the signs and symptoms. The difficulty is much greater in women, owing to the close resemblance of these masses to growths in connexion with the pelvic generative organs.

Although these growths are in the main of a simple kind the *prognosis* is far from being very favourable unless recognised and removed while of small size. A fatal result has not infrequently attended the removal of large retroperitoneal growths of this nature; or, when this has been attempted, their large area of attachment has frequently prevented any successful interference. Of the 42 cases collected by Prof. Adami (1), in 26 "the tumour was removed wholly or almost wholly. In 12, or 46·1 per cent, the operation was successful." The same writer draws especial attention to the danger due to the position of the colon in front of the tumour, and "as a consequence unless great care be taken in the

removal, the blood-supply of this portion of the intestine is cut off and gangrene or necrotic inflammation ensues, which seems to have been the history in most of the fatal cases."

Fibro-myoma. Myoma.—Rare cases of new growth originating in the retroperitoneal tissue and composed of fibrous tissue with a variable amount of plain muscular tissue, have been placed on record. They correspond in structure to the common fibro-myomas of the uterus, and with this organ some of those which extend into the peritoneal cavity are primarily connected, retaining their covering of serous membrane. In a few situations, such as the uterine ligaments and the root of the mesenteries as well as the sustentacular ligaments of the peritoneum, a small amount of plain muscular tissue is normally to be found in the subserous connective tissue, and it is from this source that these tumours arise. A large growth of this kind weighing upwards of 9 lbs. (after the escape of a large quantity of oedematous fluid) has been recorded by Mr. Sheild, and a most interesting case of multiple fibro-myomas, in a girl of nine years of age admitted to the Tokyo Hospital, Japan, is described by Anderson. In this case "twenty-one rounded tumours of various sizes, the heaviest of which weighed over 7 lbs., were removed from different parts of the subperitoneal tissue, extending from the pelvis to the hypochondrium. They were distinctly encapsulated and shelled out readily." Like the mass removed by Mr. Sheild they are described as having been "soft but elastic, white and wax-like on section." A large fibro-myoma undergoing extensive myxomatous degeneration and weighing 30 lbs. was successfully removed by Mr. Doran (15) from a woman aged 34. Of eleven cases of fibro-myoma of the broad ligament—one of the more usual situations for their development—collected by Mr. Bland-Sutton, the majority weighed upwards of 16 lbs., and all occurred in women over thirty-five years of age. So far as is known, these growths exhibit no peculiarities serving to distinguish them from the fibro-lipomas, until they are submitted to microscopical examination; and the signs and symptoms to which they give rise correspond to what has been already stated. Those situated in the broad ligament may cause considerable displacement of the uterus and ovaries, and, after remaining stationary for a long period, are apt to take on a rapid rate of growth.

Angioma.—Isolated cases of naevoid growths of the peritoneum have been recorded. Mr. Arbuthnot Lane removed a very extensive angioma from the abdominal cavity of a boy aged seven years, and Julliard a cavernous angioma weighing 8 lbs. from the mesentery of a boy who had had symptoms of acute intestinal obstruction. In Wegener's case an angioma of the ascending layer of the transverse mesocolon imitated a twisted ovarian cyst. A point of interest is the relation of these large angiomas to angio-sarcomas.

MALIGNANT GROWTHS.—**Primary.**—Malignant growths seldom arise primarily in the peritoneum. In the past numerous cases were reported

as carcinoma, and there is no doubt that microscopic sections of primary peritoneal tumours may shew a histological structure indistinguishable from that seen in carcinoma; these tumours are now regarded as belonging to the group endothelioma, and it may only be possible to prove that an alveolar tumour resembling carcinoma is in reality an endothelioma by making serial sections and so tracing the growth to its origin from a lymphatic or other vessel. Without discussing this question further, it is safe to class primary malignant tumours of the peritoneum among the sarcomas and endotheliomas, and regard the cases formerly described as carcinoma as clinically malignant, but not of the same nature as carcinoma elsewhere.

Their most usual situation is in connexion with the retroperitoneal connective tissue; less commonly in the great omentum; still less often in one of the mesenteric folds, and, very exceptionally, in the broad ligament. Mr. Pearce Gould removed a spindle-celled sarcoma weighing 21 lbs. from the gastro-hepatic omentum of a man aged 38, a most unusual position, of which he could only find one other example. In many cases the growth appears to have arisen primarily in one or other of these sites; but even then smaller masses are often to be met with apart from the main tumour, especially with those more distinctly peritoneal—the nodules being scattered about on other parts of the serous membrane and sometimes on the surface of the liver, where they are often umbilicated. In some cases there are innumerable minute growths, not unlike miliary tubercles, without any obvious primary growth, a condition of peritoneal sarcomatosis. Occasionally the retroperitoneal lymphatic glands are affected, but very rarely any other organs, whether abdominal or thoracic. In those patients in whom there is reason to believe the growth to be secondary, the testis or ovary appears to be the usual primary seat (31, 41). However extensive they may have become, even to the enveloping of such organs as the kidney and pancreas, they exhibit but little tendency to invade these viscera, although extension into the lumen of the intestine has been met with (34).

Microscopically these growths present the ordinary appearance of round-celled, spindle-celled, irregular-celled, or lympho-sarcoma, endothelioma, and perithelioma. Under the name of plexiform angio-sarcoma Waldeyer has described certain large tumours, connected with the peritoneum, consisting of new-formed blood-vessels, the outer coats of which have become the seat of extensive hyaline change. They vary much in degree of hardness in accordance with the proportion of fibroid elements and the extent of degenerative changes they may have undergone, for it is common to find in the larger masses, which may attain a bulk of 30 lbs. weight and upwards, an extensive conversion into myxomatous tissue, or even into cysts; in Dutton Steele's collection of 61 retroperitoneal sarcomas 35 per cent shewed pseudo-cysts; and reference has been already made to the association of sarcoma with fatty overgrowths. The so-called "gelatinous carcinoma" of Rokitansky belongs to this group.

Occasionally, especially in endotheliomas and peritheliomas, the mass is extensively infiltrated with pigment which is sometimes derived from blood; these neoplasms are generally very vascular and soft. The endotheliomas may present a mixed histological picture, in which the features of sarcoma and carcinoma are both present. Unlike the lipomas, these tumours are more frequent in males, and although usually met with in adults are not unknown at an early age. A doubtful history of injury is sometimes obtainable.

It is not unusual to find the peritoneum in the immediate neighbourhood of these growths exhibiting a moderate amount of chronic inflammatory change whereby adhesions of varying firmness are established between the mass and adjacent structures, especially to the parietal layer of the serous membrane; but the peritonitis is strictly limited to the parts concerned, and is quite secondary and subordinate. Contrasted with the non-malignant growths in this region the sarcomas interfere with the abdominal circulation far more seriously, as may be seen by the great distension of the superficial veins, and also by the more frequent presence of an ascites, which may be considerable, or of an oedema of one or both lower limbs.

Signs and Symptoms.—Apart from the manifestations of general malnutrition, wasting, anaemia and failing strength, which are determined by these growths wherever situated, there will be special signs and symptoms more or less distinctive in character, due to their size and position, and to this extent available for diagnostic purposes.

The retroperitoneal tumours, in their extension forwards, of necessity tend to displace the kidneys, pancreas and intestines laterally, as do the previously described solid growths in this situation; like them also they frequently retain the descending colon in close adherence to their anterior surface, a circumstance that explains the variable character of the percussion note obtainable over the most prominent part of the mass, according as the bowel is or is not distended with gas. Owing to the more extensive attachment, and the situation from which they spring, these growths are more fixed than the other varieties and move but little under manipulation. On several occasions the right kidney has been found to be congenitally misplaced, lying on the pelvic brim.

Omental sarcomas for the most part form hard, flattened, smooth, or slightly irregular masses, sometimes appearing to consist of two or more separated portions, scarcely if at all tender and seldom painful, situated mainly to one side of the middle line, and rather oftener to the right below the level of the liver from which they are more or less distinctly separated by a band of resonance due to the transverse colon. The lower margin is plainly to be felt as a thin edge, and the whole tumour, which is freely movable, especially from side to side, feels quite superficial and is uniformly dull; unless on deep percussion when a subresonant note is elicited. Such a tumour, says Sir F. Treves, "is one of the most characteristic of abdominal swellings, and feels like a cake of no great thickness moulded to the curve of the abdominal wall" (48).

Those sarcomas which grow in the mesentery, like the preceding, are very movable unless the parietal adhesions be extensive; and they preferably occupy a position in the middle line just above or below the umbilicus. In shape they tend to be globular rather than flat and, felt through the coils of intestine before they are sufficiently large to reach to the surface, they have much of the character of a tense, thick-walled cyst. Coincident with his discovery of the abdominal tumour, or preceding it, the patient may complain of vague abdominal uneasiness with a general sense of ill-health, colicky pains, nausea, sickness of an irregular character and loss of appetite, and of that feeling of weight and sinking so commonly associated with abdominal disturbance with or without a tumour. Such symptoms which are in no sense characteristic, and are apt to be set down to a gastro-intestinal indigestion, tend to progress in spite of remedies until the recognition of the tumour explains the condition. Gradually a deep-seated pain in the belly, perceptible more "from the front" than "from the back," as the patient may express it, becomes a marked feature in the retroperitoneal growths; or it may be that for this symptom relief is sought before the detection of a tumour. Later, as the growth becomes larger and more perceptible, various pressure results, especially referable to the bowels, are liable to occur; and constipation amounting to almost complete obstruction may be established.

Secondary Malignant Disease.—This may be either carcinomatous or sarcomatous, but from the greater number of primary carcinomas in the abdominal viscera secondary peritoneal growths of this character are more frequent than sarcoma. Although carcinomatous affections of the glandular structures of the abdomen are of fairly frequent occurrence, implication of the peritoneum secondary thereto is not so common as might be supposed. It is by extension of the growth from the diseased viscus, as well as by secondary infection by way of the lymphatic vessels, that the serous membrane is implicated, and probably the ovaries are the commonest source; less often the stomach, intestines, liver, gall-bladder, and pancreas, and very rarely the oesophagus or breast. Once the serosa is invaded, as Coats pointed out, particles of the tumour may be carried about to different parts of the cavity and into the subserous tissue also: the resulting growths are then really subperitoneal, and covered by an unbroken layer of serous membrane. Secondary carcinoma faithfully reproduces the microscopic structure of the primary tumour.

Frequency—Age—Sex.—Trustworthy statistics are wanting to enable any definite statements to be made as to the frequency of peritoneal malignant disease, whether primary or secondary. For lack also of precise figures the relative incidence of the disease in the two sexes cannot be positively stated, although from the frequency of cancer of the pelvic reproductive organs it would seem to be commoner among women. As regards age, it is mainly a disease of middle and later middle age, though not unknown in the young.

Morbid Anatomy.—The general appearance of growths of this character

varies much. Scattered over the peritoneum, visceral and parietal, and sometimes also invading the pleurae, are numerous small flattened nodules varying in size from a grain of sand to a pea or even larger: they are sometimes pedunculated and in clusters. As a secondary infection, according to Fenwick, this form of carcinoma is met with more often as a sequel of cancer of the gall-bladder, pancreas, or ovaries. Dr. Hebb has recorded a case in which it was secondary to a similar condition of the right pleura. Fenwick has also drawn special attention to the mesentery and mesocolon as being most affected, with the result that they become much puckered and contracted, and drag the intestines backwards towards the spinal column

In another group of cases the new growth forms large masses, the omentum being the special seat of morbid change, becoming converted into a cord-like tumour, of a variable degree of hardness, lying across the abdomen, below the transverse colon. It is seldom confined to this structure, however, numerous nodules of different size being spread about the peritoneal cavity; often also with scattered miliary formations, as already described.

A third form in which malignant disease of the peritoneum occurs is that denominated "colloid cancer." Concerning the exact nature of this disease, and especially of its mode of origin and relations, much remains to be learned. It forms the largest abdominal tumours with which we are acquainted. Prof. Osler mentions one weighing over 100 lbs. In structure the new growth, which tends to invade the entire peritoneal cavity and its contents, consists of large alveoli bounded by a connective-tissue framework, and containing a homogeneous colloid matter with occasional indications of epithelioid cells singly or in groups. While the bulk of the material is of this soft gluey character, it fills the containing spaces so tensely as to form tumours of very considerable hardness. Although some of these growths are primary and probably derived from the Wolffian body, and are endotheliomatous or myxosarcomatous, others are really secondary to carcinoma of some viscus, probably of the stomach or intestine; while the cellular constituent tissues of the growth and the stroma undergo a mucoid or colloid degeneration until spaces large enough to be visible to the naked eye are formed.

The condition of the peritoneum in these cases is noteworthy. When the growth is mainly of a disseminated miliary character the serous membrane is much thickened, sometimes to an enormous extent; partly from a diffuse infiltration of the membrane by carcinoma, and partly from an associated inflammatory new formation. Reference has been already made to the contraction of the mesenteries which may result from this thickening, and to the displacement of the intestines which ensues. Even in the absence of this condition peritoneal adhesions between adjacent viscera, or between them and the parietal layer of the serous membrane, are almost always to be found, with the result of an adhesive peritonitis determined by the new growth. The extent to which they are carried is extremely variable; sometimes they mat the various parts

together, especially in and about the pelvic cavity, into inextricable masses. Usually also there is some effusion, a few ounces or many pints; in the miliary form it is very considerable in amount. The fluid presents the ordinary characters of an inflammatory exudation or, as is often the case, it is haemorrhagic. Sometimes it has been chylous, and a remarkable case of secondary miliary carcinoma of the peritoneum, causing engorgement and rupture of the mesenteric lacteals with chylous ascites, is recorded by Fenwick. Chylous bullae were also seen in the intestinal submucosa. More often the milky character is due to fat and not to the escape of chyle. Leyden and Schaudinn described amoebic organisms in the effusion of cases of malignant disease of the peritoneum under the name of *Leydenia gemmipara*; their protozoan nature is doubtful, and this observation requires confirmation. The mesenteric and retroperitoneal glands are often unaffected, or only very slightly enlarged.

Clinical Characters.—The usual features of malignancy are exhibited in a supreme degree by most cases of peritoneal cancer; since in addition to the evil influence of the growth itself there is usually also a direct interference with the digestive and absorbent organs.

As is commonly the case in abdominal cancer, whatever the organ primarily affected, the onset is vague and insidious—a general sense of failing health and bodily weakness rather than any definite symptoms being the earliest phenomena observed. Moreover, it is practically impossible when the growth is secondary, as it usually is, to discriminate between the manifestations of the primary affection and those due to the peritoneal implication. Neither at the outset nor in the course of the disease is pain by any means a constant or often a serious symptom. Occasionally attacks of griping and colic, often accompanied by flatulence, may be succeeded by considerable intervals of complete freedom from more than a little abdominal discomfort. But, on the other hand, in some cases pain is a prominent and almost a constant symptom throughout; and the exact reason for the differences in this respect remains to be found. Tenderness also is variable in extent, but sooner or later it usually appears. The temperature, which inclines to be subnormal, may exhibit exacerbations which are attributed to a slight spread of peritonitis, especially if they occur after tapping the abdomen. All degrees of digestive disturbance are observed; the appetite is capricious or wanting, and even at first is rarely well maintained. Constipation is the rule and, determined as it is by obstructions of the intestine from the pressure of tumours and fluid, from stricture of its lumen by new growth in its walls, or from twistings and displacements due to contraction of the mesentery, is often most obstinate. Later in the progress of the case an intractable diarrhoea may set in and hasten the fatal end; or attacks of diarrhoea may alternate with constipation. Vomiting is sometimes met with. The malnutrition induced by the deficient nourishment added to the specific effect of the growth induces a weakness and fatigue marked from the first, and it is this condition, indeed, which may be the first to attract attention to the real state of the patient. Soon it is accompanied with an

increasing loss of weight, anaemia, and the invasion of a characteristic cachexia, symptoms which steadily progress.

The *physical signs* will necessarily vary with the size and situation of the growth, the presence or absence of a visceral tumour, the extent of the ascites, and the pressure effects to which these conditions give rise. When the new formations are entirely of a miliary character they cannot be detected, and their presence can only be inferred; and, since in such cases there is extreme ascites, the nature of the malady may be overlooked; but when the nodules attain an appreciable size they may be felt with varying distinctness through the emaciated abdominal wall, more especially those which have formed on the parietal peritoneum or are adherent thereto. The facility with which the abdominal contents can be palpated will necessarily depend in great measure on the amount of the ascites. The omentum, which is rarely free from the new growth, often forms the largest and best-recognised tumour; it is thickened and rounded in shape, sometimes partially broken into two or three masses, or flattened and moulded to the curve of the anterior wall of the belly. The greatest difference exists as to the range of mobility of the tumours, but their tendency is to become fixed by peritoneal adhesions. Unless the distension from fluid be extreme, when a general dull note will be obtained by percussion, the resonance over the abdomen is irregularly distributed in accordance with the size and situation of the tumour or tumours. Fenwick has particularly drawn attention to the dulness along the course of the colon in those cases in which the intestines are retracted to the spine by a thickened and shrunken mesentery; and not only are the flanks then dull, but, on placing the patient in the genu-pectoral position, the usually resonant area below and to the outer side of the kidneys is dull on one side or on both. Considerable irregularity in the contour of the surface of the abdomen is rendered more apparent by the wasted integuments. A characteristic appearance is that presented by the umbilicus, the skin around it being sometimes red and much thickened. The inguinal glands are frequently enlarged.

The duration of the disease after the existence of the growth has been recognised rarely exceeds six months, and sometimes it is much shorter; the miliary variety is especially acute in its course.

The **diagnosis** of the nature and situation of these malignant growths, whether primary or secondary, and their distinction from other intra-abdominal abnormalities are often difficult. Before the peritoneal tumours can be detected by physical examination a diagnosis of malignant disease cannot be made, though it may be suspected; especially if a visceral growth be known to exist.

The miliary form with its considerable ascites and absence of palpable tumour may be mistaken for hepatic cirrhosis, but the history of the case and the habits of the patient must be considered. In the latter affection, also, the abdominal distension is usually greater and the enlargement of the superficial veins much more marked, owing to the direct interference with the venous return.

When a definite solid tumour can be detected various are the conditions for which it may be mistaken. Of these a faecal accumulation may closely resemble an omental growth; and it may not be until after several large enemas have been administered (which in all such cases should, unless good reason to the contrary, be an invariable preliminary) that what is faecal can be distinguished from what is not. An educated touch may detect a greater hardness and uniformity in the growth than in the scybalous masses, but this quality, even if present, is not always to be relied upon, and tenderness may accompany or be absent from each disorder. In women malignant disease of the peritoneum is probably most often confounded with cystic or semi-cystic growths of the ovary or broad ligament; and a vaginal examination should not be omitted.

Both the retroperitoneal and omental growths are apt to be mistaken for renal tumours, and the relation of the dull to the resonant areas favours the error. When the mass is movable and of no great size its greater range of displacement from side to side may help to distinguish it from a movable kidney, the mobility of which is rather in an up-and-down direction; and this distinction is of service, inasmuch as the omental and mesenteric growths present in many respects a close resemblance to the dislocated viscus.

The distinction between peritoneal cancer and tuberculous peritonitis is often hard to make. There are the same fulness of the abdomen, moderate ascites, the very slight mobility of the fluid which is circumscribed by the presence of adhesions, an omental tumour, and, very commonly also, palpable nodules distributed over the abdomen. The removal of the fluid does not facilitate the diagnosis, which must depend upon other circumstances, such as the age—the tuberculous disease being much more frequent in children and young persons; the presence of tubercle in other organs; the steady progress, and, as a rule, shorter duration of the malignant disease—the tuberculous affection being often marked by periods of quiescence or even of partial improvement. A discharge of pus from the umbilicus is strongly in favour of tuberculous peritonitis; on the other hand, red nodular swellings in this locality suggest carcinoma. Sometimes tubercle bacilli or groups of cancer cells may be found in the exudate withdrawn by aspiration. Dock considered that some, but not absolute, diagnostic value may be attached to the greater number of cells shewing mitosis, which is often atypical, being found in the serous effusions associated with carcinoma than with tuberculosis. But with every care and with skilled observers the diagnosis may remain doubtful.

The *prognosis* of these tumours is invariably grave; if left alone they run a course of but a few months after their first recognition; and, unless quite small, their removal is either impossible, or the attempt will probably hasten death. Yet this is not always so; Llobet records a case of complete recovery after removal of a mesenteric sarcoma together with a considerable piece of small intestine to which it was inseparably adherent. A diminution in size has been sometimes observed after a

simple exploratory laparotomy. Cases have been recorded by Greig Smith and others in which the abdomen has been opened and an inoperable and apparently malignant tumour found, and yet complete disappearance has followed. It is probable that these tumours are inflammatory.

Anodyne applications, a judicious use of morphia internally, due attention to the bowels, and the administration of the most digestible and nutritious diet are the sole means of *treatment* at our disposal.

Cysts.—Reference has been made to the liability of certain growths, especially the sarcomas, to undergo cystic change; and this may sometimes be so extreme as to give rise to a tumour which is mainly composed of cysts, the undegenerate firmer parts forming but a relatively small portion of the mass. Besides these, however, cysts of various kinds are occasionally to be found in connexion with the peritoneum or its subjacent connective tissue; of some of these the precise nature and mode of origin are obscure, though others are sufficiently definite in character.

Hydatid Cysts.—*Vide* art. "Hydatid Disease," Vol. II. Part II., p. 976.

Dermoid Cysts.—Cysts of this class, which inasmuch as they are really teratomas are spoken of as embryomas, with their characteristic contents of fatty matter and hairs enclosed in an epithelial lined structure homologous with skin, are sometimes met with attached to the peritoneal membrane and growing into the cavity, or in the retroperitoneal tissue. The latter are probably the more frequent, and are developed in patients of both sexes behind the rectum; sometimes they grow to a great size and extend upwards behind the serous membrane as high as the umbilicus, causing considerable displacement of the adjacent viscera. As Mr. Bland-Sutton points out, those met with in this situation may be dentigerous. The intraperitoneal dermoids may be very numerous, the result of secondary infection of the membrane from rupture into the serous sac of an ovarian cyst of the same character. Such tumours rarely attain a large size, and "may take the form of minute granules on the peritoneum, each of which is furnished with a tuft of delicate lanugo-like hair, or may hang from the under surface of the liver, or form clusters like cherries upon a branch, or be embedded in adhesions between coils of intestine" (44). Such growths are exceedingly rare. Very large single cysts of this variety have also been met with containing several pounds of sebaceous matter with hair. Some of these have doubtless originated behind the peritoneum, and extended between the layers of the mesentery; but others have been found attached to the omentum, and Mr. Doran is strongly of opinion that the latter are really ovarian in origin, "but have become adherent to other structures and then separated from their pedicles" (11). Like dermoid cysts generally these tumours are very uncertain and irregular in their growth, and after years of quiescence they may increase rapidly in size. As the result of contamination from the intestine, to which they may become adherent, they are liable to suppurate: and may then burst into the bowel, the vagina, or on the surface

of the umbilicus. In rare instances these teratomas may become malignant (Montgomery).

Cysts of Doubtful Nature.—Scattered throughout medical writings are many records of cysts connected with the mesenteries, omenta, peritoneum, or subperitoneal tissue; not sufficiently numerous, however, nor described with sufficient detail to permit of exact classification or of complete recognition of their character and origin. They were frequently designated as "hydatid," a name which formerly had a wider meaning than its present restricted application, and this tends to confusion; but it is also probable that some were really sterile echinococcus cysts; these might be detected by the laminated character of the wall and the peculiar nature of the confined fluid, even in the absence of scolices and hooklets.

Other cysts of this indeterminate group probably originate from obstructed and dilated lymphatics. Such have been met with in the substance of the omentum (the other tissues of which having more or less disappeared), where they form pedunculated clusters of ovoid or pyriform bodies resembling bunches of grapes; examples of which have been recorded by Mr. Cripps and by Mr. Berry: or they may appear as elongated cylindrical swellings several inches in length and of the diameter of the finger, and contain a clear, yellowish, slightly viscid albuminous fluid. Closely similar to these, if not identical with them, are the mesenteric cysts which contain chyle. Considerable effusions of chylous fluid have been met with between the layers of the mesentery or in the retroperitoneal tissue, due probably to rupture of one or more chyliferous vessels, which form large swellings containing several pints of fluid (39). Such tumours, however, are not true cysts, since the fluid is not contained in a very definite membranous sac, but within the accidental boundaries of the surrounding tissue. Sometimes the containing sac shews traces of an endothelial lining in places, as if the cysts had originated from retention in distended lacteals or in the receptaculum chyli. By some these structures are regarded as embryonic (Fawcett). Similarly, effusions of blood (haematoma), the result of injury, may be found in the same situations.

Exceedingly thin-walled cysts, of rare occurrence in the omentum and also in the subserous tissue, are described, containing sometimes several pints of clear serous fluid. The wall itself consists of a delicate connective tissue, without any evidence of an epithelial lining. No adequate explanation has been given by the nature of these cysts, although they also have been looked upon as embryonic, and the remains of the Wolffian body has been held to be the starting-point of some which are retroperitoneal (Lockwood). Others are supposed to be developed from the lymphatic glands or vessels—cavernous lymphangioma. In some cases the cyst-wall contains plain muscular fibres, and it is suggested that such are derived from diverticula of the intestine, the communication with which has become obliterated and the mucous membrane shed. No essential difference in nature seems to exist between the serous and chylous cysts, the character of the contents being indifferent. Occasionally a

pedunculated cyst appears to have originated from one of the appendices epiploicae.

Though rare a number of *urachal cysts* have been described (14); the cyst usually lies in front of the peritoneum, but from persistence of the mesentery of the urachus the cyst may project intraperitoneally (Delore and Cotte). The "Allantoic cysts" described by Lawson Tait appear not to be of foetal origin, but to be examples of localised tuberculous peritonitis (14).

Some of the encysted collections of serous fluid which occur in the peritoneal cavity, and also (as there is good reason to suppose) in the sac of the great omentum, are inflammatory in origin and are really due to localised peritonitis, the fluid being confined by the formation of adhesions. Those met with in the lesser peritoneal cavity, often haemorrhagic in character, are usually connected with disease of the pancreas.

Clinical Characters of the Peritoneal Cysts.—The signs and symptoms of these abdominal swellings much depend upon their size and their situation. Inasmuch as they tend to interfere but little with the general nutrition, their existence is often unsuspected, and is revealed accidentally during life, or unexpectedly after death. It may be said generally that the symptoms to which they give rise are in the main due to the pressure they exert on various parts; yet even these often amount to little more than a vague sense of discomfort and weight. Some interference with the intestinal peristalsis may be induced, even to the extent of moderate obstruction, and vomiting sometimes occurs; or micturition may be rendered difficult, or too frequent, from the pressure of the tumour on the bladder. But it is seldom that a cyst, however large, leads to any ascites from compression of the portal vein, though this may follow when it is situated in the small omentum, a position in which it may also cause jaundice by obstructing the common bile-duct. When the abdominal cavity is greatly filled by many hydatid cysts, the diaphragm may be so pushed upwards as seriously to embarrass the action of the heart and lungs. Not often is pain, or even tenderness, complained of; and the presence of these tumours rarely leads to peritonitis, unless evacuation of their contents into the peritoneal cavity should occur; and not always then. The occurrence of suppuration in a cyst is usually accompanied by the general symptoms of that condition—fever, rigors, and sweating—to a greater or less extent.

Great variability necessarily exists in the *physical signs* of tumours of this class, according to their size, their situation, and their mobility. The last characteristic is best marked in the smaller and medium-sized cysts attached to the omentum or mesenteries, and it may then be manifested to a considerable degree. The special group of *mesenteric cysts* has been described as presenting the following signs: a fluctuating, very mobile tumour near the umbilicus, usually on the right side, dull on percussion except for a band of resonance over it due to the intestine, and surrounded by a resonant area. The existence of adhesions will obviously limit the range within which the swellings may be moved, and

the cysts which are retroperitoneal tend to be quite fixed. As a rule it is rather in a side-to-side direction than from above and downward that the mobility is greatest. The feel of the tumour is tense and elastic, but it is not always easy to distinguish a cyst from a solid growth, especially if it be deeply situated. Fluctuation is perceptible when the growth is large, and the characteristic "fremitus" may be elicited when a hydatid is near to the surface. The cyst may appear smooth and uniform, or nodular and irregular, the latter being generally the case with hydatid tumours.

The *duration* of these various forms of cysts is most uncertain and difficult to ascertain. They have been met with in infants and in young children, as well as in adults. The time of their onset can scarcely ever be known; but as contrasted with most of the solid tumours of this region, the cystic swellings are of far longer duration, and indeed may remain with little or no change for years. They are, however, liable to suppurate, and then to give rise to more active and serious symptoms; or they may burst either into the peritoneal or pleural cavities and set up grave mischief of a secondary character.

The *diagnosis* of these cysts is often very difficult. Those which are deeply situated behind the peritoneum may be easily mistaken for solid growths in that situation, the fluid character of the contents not being recognisable, even if there be no undue thickness of the abdominal walls; those which originate in the pelvis, or dip down into it, may be excusably confounded with cysts of the ovary or its appendages. When, however, the tumour is single, and its cystic characters are recognised, it may still be impossible to assert that it is hydatid in nature. It is commonly recommended to puncture the swelling and to examine the fluid withdrawn; but apart from the fact that a procedure apparently so simple is not wholly unattended with danger, grave symptoms and even death having followed from an exploratory tapping, it is, in my opinion, better to remove the tumour at once; it is always liable to increase in size and to become troublesome, and, when free from adhesions, the operation is accompanied with a minimum of risk.

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SUBPHRENIC AND OTHER FORMS OF PERITONEAL ABSCESS

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Two distinct forms of Subphrenic Abscess are met with clinically. Those which contain pus only, and those which contain gas as well as pus—Subphrenic Empyema and Subphrenic Pyopneumothorax. The term subphrenic abscess belongs rather to clinical medicine than to the more exact nomenclature of systematic pathology; but it is analogous to other terms which denote localised peritoneal inflammation, of which the most familiar are pelvic, appendicular, pericolic, perinephric abscess. It is, however, even less precise, since subphrenic abscess may probably be due to a greater number of causes than any of the others, and the symptoms to which it may give rise not only differ widely, according to the position and extent of the lesion, and the nature of the exciting cause, but also owing to the frequency with which thoracic complications occur. The term is nevertheless sanctioned by usage, because it expresses a pathological complex, which is of unusual interest from the clinical standpoint,

even though it gives no precise indication as to the real nature of the affection in any particular case.

History.—The more accurate knowledge of the conditions which lead up to subphrenic abscess and the symptoms which may be expected to arise, seem to date from the year 1845, when G. H. Barlow published a paper "On Perforation of the Stomach, with Obscure Thoracic Symptoms," but the importance of the subject received little recognition until the appearance of von Leyden's classical paper (40), since which time a great number of cases have been recorded, and the main symptoms and pathology of the condition have received general recognition.

Causation.—With the exception of certain cases of chronic peritonitis, due to pressure or some other form of non-bacterial irritation, it is probable that all cases of localised peritonitis, and certainly all cases of peritoneal abscess, are excited directly or indirectly by one of the pathogenetic micro-organisms, and that the so-called "chemic" cases (which are not experimentally produced) are due primarily or secondarily to bacterial invasion. For this reason I have not adopted Nothnagel's classification of the causes of peritoneal abscesses into bacterial, "chemic," and mechanical. With regard to this particular point, it has been shewn (23) that the absorption from the healthy peritoneal surface is so rapid that the absence of bacteria from the peritoneal fluids cannot be taken as positive evidence that they have not been the exciting cause of an effusion. Organisms capable of exciting inflammation have been found by Dr. Durham in the omentum and anterior mediastinal glands although they were absent from the peritoneal exudate. Messrs. Dudgeon and Sargent support this view (22B, p. 26), and arrive at the conclusion, after an exhaustive inquiry, that there is not sufficient evidence to justify the opinion that chemical peritonitis exists as a clinical entity apart from bacterial infection. If these views be correct, the causes of subphrenic abscess and other forms of localised peritonitis and peritoneal abscesses may be grouped under two headings: (*a*) Those which are primarily mechanical; (*b*) those which are primarily bacterial.

The bacteriology of peritonitis is fully considered elsewhere (p. 895), but here it may be incidentally stated that between twenty and thirty different species of micro-organisms have been found in acute peritonitis.

Etiology.—Local peritoneal abscess may result from a great number of causes in the abdomen, and also from some in the thorax; whilst the tracks by which pus may spread from the initial lesion, until it finds its way blocked by the diaphragm, must sometimes be very devious. Subphrenic inflammation may, a priori, arise from a lesion in any abdominal viscus, however remote from the diaphragm; in practice it is found that the great majority of cases arise from perforated gastric and duodenal ulcers, appendicitis, or suppuration in and around the liver. Of 106 cases collected by Maydl in 1894, 20 were due to lesions of the stomach, 14 to lesions of the appendix, and 30 to affections of the bile-ducts. Prof. Leith found that out of 212 recorded cases 74 were due to gastric ulcer, 37 to affections of the liver, and 20 to

appendicitis. Prof. Osler quotes 142 cases, of which 56 per cent were due to gastric ulcer, 10 per cent to appendicitis, and 6 per cent to duodenal ulcer. No useful purpose would be gained by multiplying these figures. They shew that a large proportion of the cases of subphrenic abscess arise from gastric ulcer, and that considerably more than half of all cases arise from lesions of the digestive tract and liver. Messrs. Wallace and Sargent have tabulated 1075 cases of appendicitis, amongst which there were 7 cases of right-sided subphrenic abscess, and Elsberg gives records of 73 cases of subphrenic abscess resulting from appendicitis, of which 27 per cent were extraperitoneal, 25 per cent perforated the diaphragm, and 15 per cent contained gas.

As regards the relative frequency of occurrence, Christian and Lehrs' observations are of interest. Amongst 4028 autopsies after death from all causes they found 86 cases directly or indirectly due to appendicitis. In 7 of these there was either subphrenic abscess or some closely allied condition; the abscess was on the right side in 6 cases. In the remaining case there was a left-sided subphrenic abscess and left-sided pleurisy. The occurrence of a subphrenic abscess and pleurisy on the side opposite to the initial lesion is not very uncommon. Other cases are recorded by Coates and by Stewart. These figures, taken from a great number of cases, indicate the relative proportion of deaths from subphrenic abscess as compared with all other causes. In Lang's 173 cases (8) thoracic complications were present in 140; pleurisy (fibrinous and sero-fibrinous) in 41; empyema in 16; pericarditis in 16; adhesions between lung and diaphragm and ulceration through diaphragm in 7; perforation into the lung in 34; into the pleura in 23; and into the pericardium in 3. In Elsberg's 73 cases, perforation of the diaphragm occurred in 18, or nearly 25 per cent.

The anatomical conditions which decide the course which pus may take from any focus of abdominal suppuration, supposing that the normal relations are not disturbed, have been fully studied by Dr. Box and Mr. Eccles, who give a diagram indicating the common positions of intraperitoneal abscesses and the directions in which such abscesses tend to track. The normal relations are, however, frequently altered by adhesions, and the pus-track may be directed out of its path, so that the abscess resulting from a left-sided gastric ulcer may even track to the right side, and penetrate into the right pleural cavity (31, 38), whilst appendicitis may give rise to a left-sided subphrenic abscess (14, 20). Clinically it is found that suppuration starting from the appendix tends to track up between the colon and the postero-lateral abdominal wall, and to form an abscess in the region of the right kidney, or to spread up between the liver and the diaphragm. Even suppuration of pelvic origin may spread in the same way along the ascending or descending colon. If it is not obliterated by adhesions before perforation occurs; the lesser sac of the peritoneum may be the seat of a collection of pus, resulting from some lesion of stomach, duodenum, pancreas, liver, or left kidney.

Age.—Although subphrenic abscess may occur at any age, it is more common in adults than in children, yet Jopson operated on a case in a child fifteen months old, in which the abscess pointed externally between the 9th and 10th rib in the mid-axillary line.

Location of Subphrenic Abscesses.—The common situation of subphrenic abscesses on the right side is between the liver and the right cupola of the diaphragm, and on the left side between the spleen or stomach and the left cupola; a subhepatic abscess is most frequently found in the right kidney pouch. The right- and left-sided abscesses may be defined by the falciform ligament which is attached to the under surface of the diaphragm, near the right limit of the pericardium, and to the anterior abdominal wall an inch or so to the right of the middle line; but in any given case it may be impossible to recognise such delimitations clinically, either because the boundaries have been broken down by suppuration, or because the physical signs are due to collections of pus on both sides of the falciform ligament, as has in some instances been found.

Clinical Course.—*Onset.*—The very various pathological conditions included in the generic term "subphrenic abscess" cannot be expected to present a uniform clinical picture. The course of the disease in fact varies as widely in different cases as do the associated visceral lesions, and the micro-organisms by which the suppuration is excited. In one case the onset may be insidious, the progress slow, the resulting symptoms ill-defined or difficult of interpretation, and the course essentially chronic, whilst in another there may be the sudden catastrophe which follows perforation of one of the hollow viscera into the peritoneal cavity, with easily recognisable symptoms and physical signs, resulting it may be shortly in collapse, and, if unrelieved after a few hours, in death. Again, in one class of cases there may be well-marked symptoms of the exciting cause, such as in the abdomen—a previous appendicitis, duodenal or gastric ulcer, or dysentery; in the thorax—empyema, or some other acute local inflammatory condition. For these reasons no attempt will be made to sketch a case which may be taken as typical. Each case must be judged on its own merits, having due regard to the history and method of onset, as well as to the symptoms, and physical signs which it presents. These will be discussed later on.

Course and Progress.—The progress of the disease, like its onset, is determined partly by anatomical and partly by pathological conditions. Thus, the result of the inflammation will vary according as it is intra- or extra-peritoneal. There may be adhesive inflammation, diffuse suppuration, localised abscess, or suppuration combined with the formation of gas. The suppuration may spread by way of the lymphatics, by metastasis, or by actual continuity. It is found clinically that extraperitoneal suppuration tends more easily to perforate the diaphragm into the pleura, whilst an intraperitoneal subphrenic abscess spreads into the thorax more slowly, and often ruptures directly into the lung, on account of adhesions formed between the lung and the diaphragm before the rupture occurred. Adhesions between the lung and diaphragm, or perforation into pleura,

lung, or pericardium, were found by Lang (*vide* p. 997) in 67 cases out of 173 (8), or nearly 40 per cent.

The course of the disease may be acute or chronic. Cases have been under my care in which the symptoms have been present from a few days to two or more years. In Messrs. Wallace and Sargent's 1075 cases of appendicitis in St. Thomas's Hospital, 1893-1903, subphrenic abscess was detected in seven instances, at intervals varying from two days to seven weeks after laparotomy. It may be said that as a rule the more chronic the onset the more likely is the suppuration to be limited by adhesions, and in so far to be proportionately amenable to treatment, or to spontaneous evacuation.

Methods of Spread.—All "pus" infections may spread from the initial lesion by the blood-stream, by the lymphatics, and by direct continuity, but in addition to these there are some special factors which determine the spread of pus within the abdominal cavity. The most important are:—(1) The seat of the initial lesion. (2) The normal anatomical boundaries formed by the mesentery, mesocolon, falciform ligament of the liver, and the spinal column. (3) Gravitation. (4) The movements of the diaphragm. The first of these needs no comment. The anatomical relations have been well studied by Dr. Box and Mr. Eccles and also by Dr. Box and Dr. Russell. As has been said, it is found that extraperitoneal suppuration, such as may result from appendicitis, tends to track up between the colon and abdominal wall, and burrow between the liver and diaphragm, often infecting the tissues of the diaphragm, and perforating into the pleural cavity or lung. Intraperitoneal suppuration from the appendix, pylorus, or duodenum tends to gravitate towards the right kidney, and to form an abscess in the right kidney pouch, directed to it by the prominence of the lumbar spine and by the inclination of the transverse mesocolon. Inflammatory lesions of the gall-bladder and bile-ducts not infrequently give rise to abscesses in the same situation (4). There is a similar tendency for pus to collect on the left side in the left subphrenic region, which contains the spleen and the contiguous portion of the stomach (4, p. 383). The other factors mentioned above which determine the direction ordinarily taken by pus do not need detailed consideration.

The direction in which subphrenic suppuration spreads is generally upwards, so that it is much more likely that the pleura or lung will be infected from below the diaphragm than that the peritoneum will be infected from the thorax. Invasion of the abdominal from the thoracic cavity does, however, occasionally occur apart from a general streptococcic or pneumococcic infection, as shewn by several cases under my care in which the diaphragm has been infected from above by empyema or bronchiectasis. Many other pathological conditions, such as pulmonary abscess or gangrene, tuberculous lesions, purulent effusions in the pericardium, are known to give rise to subphrenic inflammation (47), but from their rarity they are not of much practical importance, although the possibility of their occurrence must be remembered. In

rare instances suppuration may spread upwards from the abdomen with apparent disregard of anatomical boundaries. Thus, in one case under my care an abscess in the right kidney pouch penetrated the liver, and formed a long sinus which joined two abscesses, one subhepatic and the other subphrenic.

Spontaneous Termination.—Localised subphrenic inflammation may terminate, just as other forms of peritonitis, either by the destruction of the infective agent and the absorption of the infective products, or by the formation of more or less dense fibrous adhesions which limit the suppuration. If pus form, the abscess, unless operated on, may eventually rupture externally, into one of the neighbouring viscera or serous sacs, or into the general cavity of the peritoneum. A case of the first kind was under my care; the patient gave a history that 21 months previously he had been shot. The bullet had penetrated the stomach, diaphragm, and lung, infecting its course with the stomach contents, and setting up an adhesive inflammation between stomach, diaphragm, and left lung, so that there was a dense mass of organised adhesions both above and below the diaphragm, eventually resulting in bronchiectasis.

In those cases in which spontaneous evacuation takes place, the rupture may occur, as stated above, into the pleura, lung, bowel, through the integuments, or into the peritoneal cavity or pericardium. I have seen one case resulting from injury in which the abscess pointed in the epigastrium and ruptured also into the lung. The patient (a boy) eventually recovered. In another an hydatid cyst ruptured into the lung, and was opened in the left hypochondrium, so that there was a sinus from the abdominal wall through the pleura into the air-passages.

Symptoms.—The symptoms of subphrenic abscess are as variable as the causes which produce it and the circumstances in which it may be developed. On the one hand, they may be those of some acute abdominal disease, and on the other, it may appear as though the thoracic organs were mainly invaded. The temperature may vary from the high fever of an acute purulent infection to that of the subnormal temperature of collapse. Similarly, the pain and distress may be very slight (as is not infrequently the case in acute perforative peritonitis, owing to a virulent form of septic intoxication). On the other hand, the pain may be intense—almost unbearable—when the nerve-endings in the diaphragm or pleura are implicated, although the condition of the patient may not be so critical as it is in some of those cases in which the pain is almost a negligible quantity.

It may, in fact, be said, that but little reliance should be placed on any one symptom. A rational diagnosis can only be made by weighing the physical signs in conjunction with the evidence of some pre-existing, disease, *e.g.* gastric or duodenal ulcer, appendicitis, or one of the other lesions known to be a likely point of origin of some inflammatory process. On these grounds the physical signs must be carefully considered in some detail.

The physical signs of subphrenic abscess may very closely simulate two

widely different classes of lesions according as the abscess cavity contains pus alone, or pus and gas. Some writers seem to assume that the characteristic signs of a subphrenic abscess are those of a gas-containing cavity. This is in reality far from being the case. In a considerable majority of instances the physical signs are those of a collection of fluid between the lung and diaphragm, or between the diaphragm and abdominal cavity, or even of a solid body in one or other of these situations, rather than those of the so-called subphrenic pyopneumothorax. Elsberg examined 73 cases of subphrenic abscess resulting from appendicitis, and found 15 per cent, or only 1 in 6·6, which contained gas. The difficulties in the way of accurately localising a subphrenic abscess which contains gas are far less than when there is pus only. *Cases in which there is no formation of gas most nearly resemble empyema*, and the conditions are often so obscure that they are frequently (even at the operation) mistaken for that affection, even though a previous history of gastric or duodenal ulcer, dysentery, or attacks of biliary colic, may have suggested that there was a collection of pus below the diaphragm. Examination by the *x*-rays in such cases might reveal the position of the diaphragm, and give invaluable assistance in localising the lesion. These points will be more fully considered under diagnosis.

The effect of an uncomplicated right-sided subphrenic abscess is to raise the right dome of the diaphragm and to depress the liver (4, p. 385), whilst a large abscess below the liver, and occupying the right kidney pouch, may give rise to similar physical signs. In both cases the diaphragm may be raised, but when the abscess is below the liver, the edge of the liver would be raised instead of being depressed, as it would be if the abscess were situated above it. These distinctions may, however, be entirely nullified by the fact that the lesion which has given rise to the subphrenic abscess may have also caused enlargement of the liver, as well as adhesions, by which the normal movements of the liver and diaphragm are impeded, or even entirely prevented. Where the collection of pus is sufficiently large to be mapped out by percussion, it is generally stated that the contour of the upper limit of percussion dulness gives valuable assistance, since it is definitely rounded, inasmuch as it follows the outline of the diaphragm. In some cases this may be so, but in a large proportion the actual outline of the subphrenic collection is masked by a pleural effusion, which has resulted directly from subphrenic infection. In Lang's series of 173 cases (8) the pleura was affected no less than 80 times. There is reason to suspect that a good many cases in which a superficial collection of serum has been found, overlying a more deeply seated collection of pus, during an operation for suspected empyema, are in reality cases of subphrenic abscess, and not of empyema. At any rate, this possibility should be borne in mind when such a condition is met with. No confident deduction, however, can be made from the isolated observation that a layer of serum is found overlying a collection of pus. I have had under my care a patient with three separate layers of fluid in the thorax—serum, sweet pus, offensive pus—all at the

same time. A case has come under my notice with a history of slight gastric disturbance and haematemesis, followed by a left-sided empyema, which was operated on. Subsequently, relying on the convex outline of an area of dulness on the right side of the thorax and the slight displacement only of the heart to the left, I surmised that there was probably a collection of pus below the diaphragm. It turned out that the pericardium had been fixed by the old left-sided empyema, and so the heart had been prevented from shifting towards the left, and the dulness on the right side was in fact due to a pyopericardium with its outline rounded off to the right by pleural effusion. This case is a good illustration of the difficulty of arriving at a correct solution of the problem by physical signs alone. In another case admitted under my care for an acute pleural effusion on the right side (which was in fact present), the pleural effusion was secondary to a large subphrenic abscess.

Local bulging over the lower part of the thorax in cases in which there is ground for suspecting some deep-seated lesion in the neighbourhood of the diaphragm is, as far as it goes, in favour of the lesion being subphrenic, since in empyema there may be a considerable collection of fluid, with little increase in girth. In fact, the girth in old-standing chronic cases of pyo- or hydro-thorax may even be diminished on the affected side, and if the girth be increased, the increase is as a rule not local but general. It is only in very rare instances that chronic encysted empyema in the young causes local alteration in the curvature of the ribs.

If there were any means besides the *x*-rays by which the position of the diaphragm could be determined with certainty, the differential diagnosis between supra- and sub-phrenic collections of fluid would be greatly simplified. Litten (42, 43) has described a wavy or shadowy line running over the chest wall during respiration, which he considers to mark the position of the diaphragm as it contracts.[1] I have not been able to satisfy myself as to the constancy or importance of this sign in inflammatory lesions in the immediate neighbourhood of the diaphragm. Even if it were always visible in the healthy individual, it is certain that in a great number of cases of suppuration, either below or above the diaphragm, the movements of that muscle are so much interfered with by adhesions or restrained by pain, that this sign is deprived of much of its value, just in those cases in which it would prove most useful.

On the Simulation of Acute Peritoneal Inflammation by Diseases of the Pleura, Lungs, Pericardium, and Heart.—Subphrenic abscess, if there is no formation of gas, may be mistaken for some acute inflammatory lesion in the thorax, and vice versa, especially if the pain be great and the physical signs indefinite. As is well known, the pain of pneumonia is frequently referred to the upper segment of the abdomen; it may be accompanied by vomiting and pyrexia, and if the pleural surface of the lung be implicated, there may be lessened mobility of the side, owing to

[1] A succinct and clear account of the method of observing this phenomenon is given in Brown and Ritchie's *Medical Diagnosis*, p. 228. Their diagram is taken from Zadel, *Das Spiel des Zwerchfells*. Berlin, 1906.

the movements being restrained on account of the pain. In these acute cases in which the physical signs are not conclusive, and there is no history pointing to abdominal disease, the catchy character of the respiration, the alteration of the pulse-respiration ratio, the superficial tenderness over the skin of the abdomen, not increased by firm pressure, and herpes on the lips, would be in favour of pneumonia. A history of previous vomiting, pain after food, extreme tenderness on deep pressure, obliteration of the lower segment of liver dulness, free fluid in the flanks, and the other well-known signs of perforative peritonitis, are, if present, positive evidence in favour of the lesion being abdominal; but when they are absent it may be difficult to decide on which side of the diaphragm the lesion is situated. For this reason caution is necessary in drawing deductions from evidence of pleural effusion after the occurrence of abdominal symptoms, since the combination may occur in inflammatory affections on either side of the diaphragm. It is of great importance to recognise the frequency with which abdominal pain occurs in intrathoracic disease, since the pain is not infrequently associated with disturbance of the digestive organs, such as vomiting, diarrhœa, and distension of the stomach, so that, in the absence of physical signs, the clinical conditions may closely simulate some perforative lesion in the posterior wall of the stomach or duodenum. Cases are on record (cf. 3, 51) in which an abdominal operation has actually been performed when the lesion was above the diaphragm, and in many the decision has hung in the balance. Several of these latter cases have come under my own observation.

The pain of an intrathoracic lesion referred to the epigastrium by the 7th, 8th, and 9th dorsal nerves on the left side may give rise to the suspicion of a perforated gastric ulcer; or if the inflammation be on the right side, some lesion of the liver or gall-bladder may be suspected. When the 10th dorsal nerve is involved the pain simulates that of acute peritonitis. Lastly, the connexions of the three splanchnic nerves with the rami communicantes of the 5th to the 12th dorsal ganglia, explain the acute digestive disturbances which not infrequently accompany the pain of acute intrathoracic disease. As a general rule pain referred to the abdomen from intrathoracic disease is associated with more or less acute superficial tenderness, rather than with pain which is greatly aggravated by pressure. Agonising pain on pressure so frequently accompanies acute abdominal disease that its presence should always excite suspicion, even if there are at the same time evidences of pleural or pulmonary inflammation.

Sufficient has been said to shew some of the difficulties which may arise in attempting to differentiate the symptoms of acute thoracic from those of acute abdominal disease when the lesion is in the immediate neighbourhood of the diaphragm. The main points which should arouse suspicion that a case is thoracic and not abdominal are:—(i.) That the pain referred to the abdomen in acute pulmonary and pleural inflammations is mainly superficial, and is not increased by deep firm pressure. But it is necessary to be cautious, since in acute purulent peritonitis the

patient may complain of little or no pain, and sometimes tolerates free manipulation without flinching. (ii.) That there is no positive indication of intra-abdominal disease. (iii.) That the tongue is fairly clean, the pulse not that of collapse, the aspect that of suffering only, and not of acute abdominal disease. (iv.) That the respirations, which in acute pulmonary or pleural disease are increased disproportionately to the temperature, will probably be jerky and accompanied by the well-known pneumonic grunt. Attempts will be made to restrain the lower thoracic rather than the abdominal movements, whilst the abdominal muscles, although held rigid, may be relaxed during inspiration.

On Gas-containing Subphrenic Abscess.—*Subphrenic Pyopneumothorax.*—Gas may originate in a subphrenic abscess in several ways. In a considerable proportion of cases it is present as the direct result of a perforation of one of the hollow viscera, most often of the stomach or duodenum. In such cases the gas is derived not only from the perforated organ, but also from the action of micro-organisms. Mr. Dudgeon isolated the *Bacillus coli* and *Staphylococcus cereus albus* from two cases of gas-containing subphrenic abscess (22B, p. 95); and besides these organisms two other species of cocci and bacilli, besides *B. proteus vulgaris*, were present. He also informs me that he has found *B. aerogenes capsulatus* is an abdominal lesion without any gas formation, though it is generally stated that this bacillus is an exciting cause of the gas found in peritoneal abscesses. A direct perforation of the wall of the diseased organ does not appear to be a necessary precursor to the formation of gas, which may arise solely from fermentation set up by bacteria in the closed sac of the peritoneum. Atmospheric air may of course be admitted to an existing abscess by its rupture externally, or into one of the air passages, or by an operation wound; but from the point of view of diagnosis such cases are of secondary importance, since the circumstances in which they arise almost preclude any doubt as to the nature of the lesion.

Physical Signs and Clinical History.—The physical signs and clinical history of a gas-containing subphrenic abscess—the Pyopneumothorax subphrenicus of Leyden—differ very widely from those found in cases in which pus alone is present, and they deserve special consideration. The diagnosis of the anatomical position of gas-containing cavities below the diaphragm, although it presents considerable difficulty, is easy compared with the uncertainty which attends the differentiation of a subphrenic abscess (which contains fluid only) from a basic empyema, or some other lesion either immediately above or below the diaphragm. The presence of a gas-containing cavity in contact with the walls of the thorax is easily recognised if the characteristic physical signs are present. As these physical signs will be fully discussed under Pneumothorax (*vide* Vol. V.), it is unnecessary to give any detailed description of them here. It will suffice to mention them in the order of their diagnostic value.

(i.) Coin percussion note. (ii.) Increased percussion resonance on the affected side with increased girth. (iii.) Displacement of organs away from the affected side. (iv.) Succussion splash; metallic tinkling and echo; distant metallic respiratory and heart sounds, diminished breath-sounds. (v.) If fluid be present as well as gas, impairment of resonance on percussion below the area of the metallic note which shifts with the position of the patient.

All these physical signs may be found in pyopneumothorax, gas-containing subphrenic or hepatic abscess, suppurating hydatid cyst, dilated stomach (especially when associated with pneumonia), or a stomach which has entered the thoracic cavity through a ruptured diaphragm, and possibly also in pneumopericardium. There is only one other condition which might possibly give the same physical signs, namely, the complete excavation of the whole of one or more of the lobes of the lung, such as, in rare instances, is found in tuberculosis. It is difficult, and I believe sometimes impossible, to be certain as to the differential diagnosis between an enormous cavity in the lung, and a limited pneumothorax above the diaphragm;[1] but it is hardly credible that such a lesion could be mistaken for a *subphrenic* pyopneumothorax, since complete excavation of the lung cannot arise without evidence of advanced and probably long-continued disease. The real difficulty arises in deciding, with certainty, whether a gas-containing cavity external to the lung is above or below the diaphragm. Little reliance can be placed on the physical signs alone in differentiating between a sub- and supra-phrenic pyopneumothorax, since, as far as I am aware, there is no physical sign which occurs in pneumothorax which may not equally occur in gas-containing cavities below the diaphragm. In the case of pneumothorax the coin percussion note hardly ever extends beyond the limit of the ribs, except in the very rare cases of inversion of the diaphragm; so that if the coin percussion note extends over the upper part of the abdomen, as well as over part of the thorax, there is prima facie ground for the conclusion that the cavity is subphrenic. Again, as far as I know, the hyperresonance of a subphrenic cavity never extends to the extreme apex of the lung, so that if the physical signs of an air-containing cavity are found over the upper third of the thorax, it is practically certain that the lesion is intrathoracic and not abdominal. Pneumothorax, both subphrenic and supraphrenic, may, in rare instances, come on insidiously.[2] In such cases neither the physical signs nor the mode of onset necessarily give any conclusive evidence as to the site of the lesion. These questions are further considered under the next heading.

Diagnosis.—From the foregoing account it is evident that the diagnosis of a subphrenic abscess must sometimes present great or even insuperable difficulties. Those abscesses which do not contain gas are liable to be

[1] If the bronchus leading to a massive excavation in the lung is blocked, the physical signs may, I believe, be identical with those of a pneumothorax; but I have never heard a succussion splash in a cavity however large, although Laennec speaks of its occurrence.

[2] While writing the above, a gentleman consulted me with a large pneumothorax, which had come on without any marked symptoms, and without any great distress.

mistaken for empyema, for a solid tumour, or for a collection of fluid between the base of the lung and the diaphragm; whilst those which do contain gas, although more easily recognised, very closely resemble pneumothorax, or one of the gas-containing cavities situated in the diaphragmatic zone. In either case the clinical history is often the most important factor in arriving at a correct diagnosis, and should be closely examined for evidence of any of those lesions in the abdomen which are known to lead to suppuration. Of these the most important are ulcerative or perforating lesions of the stomach and bowel, including appendicitis, lesions of the gall-bladder and ducts, and even in rare instances bacterial infection apart from any gross pathological lesion. Suspicion of a subphrenic abscess should be excited in any case in which the symptoms and antecedent conditions render such an explanation probable. This is specially so in those cases in which (although the ordinary evidences of intrathoracic disease are absent), percussion resonance is found to be impaired over the bases of one or both lungs, with diminished vocal fremitus and respiratory murmur. Further, local bulging of the lower ribs, without obvious displacement of the heart to the opposite side, if associated with irregular pyrexia and leucocytosis, would lend probability to the surmise that some deeply seated inflammatory lesion was present. When a collection of pus below the diaphragm is large, it may be found limited by the falciform ligament, and so give indications of its position; but the information which might be gained in this way may be neutralised by the occurrence of an abscess on both sides of this boundary (cf. Hunt (34)).

If no information can be elicited from the physical signs in the abdomen, attention must be turned to the condition of the thorax, and the evidence as to the probability of previous pulmonary or pleural disease critically considered. In this connexion the examination of the sputum may give valuable information. The presence of tubercle bacilli in a case in which the onset has been sudden, and there has been long continued cough and purulent expectoration, is *ceteris paribus* conclusive evidence in favour of pneumothorax. If the pus discharged by the mouth has a faecal odour, or contains liver-cells, it is practically certain that the lesion is subphrenic. The horribly fetid sputum of bronchiectasis does not give much help in the diagnosis, as this condition may be either the cause or the result of a subphrenic abscess. A collection of pus in the mediastinum or pleura, which is discharging through the lung, will in many instances give rise to profuse purulent expectoration without odour and free from blood, and in so far is evidence that the lesion is above the diaphragm; but these distinctions must not be too closely pressed, since purulent expectoration from below the diaphragm is sometimes odourless, and pus from an empyema which has ruptured into the lung is not infrequently very offensive.

The difficulties which must often arise in the diagnosis of purulent collections beneath the diaphragm have been incidentally pointed out in considering the physical signs. Too much stress cannot be laid on the

possibility that physical signs, which might result from an uncomplicated collection of pus below the diaphragm, may be and in a large number of cases are obscured, or even entirely obliterated, by effusions—serous, purulent, or haemorrhagic—into the pleural cavity above them; whilst such information as might be derived from bulging of the parietes, depression of the liver, and so forth, is too uncertain to be relied on in those cases in which trustworthy evidence is most needed.

As has been previously insisted on, many different pathological conditions may give the same physical signs, and even the same symptoms as a moderate collection of pus immediately below the diaphragm, whether situated in the neighbourhood of the liver, or of the stomach. Of these the most commonly found below the diaphragm are hepatic abscess, hydatid cyst, actinomycosis, or even primary carcinoma of the liver, when accompanied with marked pyrexia, as it sometimes is; above the diaphragm—empyema, neoplasm, and chronic inflammatory lesions at the base of the lung. If the history of the case is indefinite and the onset insidious, it may be impossible to decide with any certainty what is the precise nature, or even the situation of the lesion, until an exploratory operation has been undertaken. Even then, as has been pointed out, it is not always easy to say definitely on which side of the diaphragm the collection of pus is situated. To these difficulties must be added those which arise from the simultaneous occurrence of symptoms of thoracic disease, such as pneumonia or pleural effusion, the physical signs of which may completely mask the conditions which underlie them. In weighing the evidence it must also be taken into account that a considerable period may elapse between the initial lesion and the development of symptoms which suggest the presence of suppuration in the neighbourhood of the diaphragm. This period may extend to some months, or even possibly to a year or more, and it not infrequently happens that the collection of pus is separated entirely from the primary seat of infection, as in abscesses round the right kidney, the result of a previous appendicitis. A history of a recent abdominal lesion with physical signs of some pathological condition in the zone which lies intermediate between the thoracic and abdominal organs, or an undue fulness round the right kidney, especially if accompanied at the same time by an irregular pyrexia, should suggest the possibility of some deeply seated inflammatory lesion below the liver or diaphragm.

Clubbing of the fingers, as has been said, occurs in recent cases of bronchiectasis and in chronic empyema. As far as I am aware it has never been observed in subphrenic abscess, however chronic, unless the lung has been secondarily affected. For this reason, if present, it is prima facie evidence that a lesion giving physical signs at the lower part of the thorax is above rather than below the diaphragm. In a case under my care in which the fingers were clubbed, an abscess was confidently believed at the operation to be below the diaphragm, but at the autopsy it was found that the lesion was a chronic empyema with fibrosis of the base of the lung.

It might be supposed that valuable information, as to the cause of the dulness on percussion over the base of the lung, would be elicited by observing the movements of the edge of the lung, when mapped out by percussion during inspiration and expiration. If the collection of fluid responsible for the dulness on percussion be below the diaphragm, the edge of the lung is said to descend over and encroach upon the area of dulness during inspiration, and recede during expiration. This may occur when no adhesions are present to interfere with the movements of the diaphragm or lung; but, as has been pointed out, the movements of both chest and diaphragm, and consequently of the lung, are often greatly restricted in subphrenic abscess. When from the physical signs it is doubtful whether a pneumothorax or a gas-containing subphrenic abscess is present, this movement of the lung may be of some service in helping to locate the lesion, since it is unlikely that the edge of the lung will descend during respiration in pneumothorax, since the line of hyperresonance as a rule remains unaltered during respiration. On the other hand, if there are no adhesions, and the muscle substance is not infiltrated with pus, the diaphragm may move downwards when the lesion is below it, though in inflammatory conditions such as those under consideration it is very unlikely that the relative mobility of the parts will be preserved. The tissues in the majority of cases are matted, and the contiguous organs bound down by adhesions.

As has been pointed out in discussing the physical signs, the diagnosis of a gas-containing subphrenic abscess is often much more easy than when pus alone is present. The extension of the coin percussion note, coupled with amphoric breath-sounds and heart-sounds with a musical timbre, beyond the limits of the thorax, is almost conclusive evidence that the condition is subphrenic and not intrathoracic. There are, however, rare instances in which the diaphragm is inverted; this physical sign might then be misleading: but such cases occur mainly, if not solely, after long-continued pulmonary or pleural disease, and are therefore little likely to be mistaken for a subphrenic pneumothorax. The same may be said of those massive excavations of a complete lobe of the lung which sometimes occur. This condition may be indistinguishable from pneumothorax by physical signs alone, especially if a bronchial fistula has been established, though in the case of the pulmonary excavation the contraction of the side will almost certainly be pronounced; whilst in a hydropneumothorax, even after the lapse of very considerable periods, the girth of the affected side may remain larger than normal. The problem of the diagnosis between these conditions and subphrenic abscess could hardly ever arise. The same may be said of some of the other lesions which give the physical signs of a gas-containing cavity beneath the chest wall, such as pneumopericardium, or a hernia of the stomach through the diaphragm. If the existence of such conditions is borne in mind, the circumstances in which they originate almost preclude any real difficulty in diagnosis. A case has, however, been recorded (21) in which what was thought to be a subphrenic pneumothorax turned out to be a local distension of the

colon, which when examined with the *x*-rays gave the appearance of a gas-containing subphrenic cavity. These and similar cases are too rare to need more than a passing mention.

On the other hand, an acute distension of the stomach, which is sometimes found in certain diseases of the base of the lung, may give rise to symptoms very closely resembling those found in sub- or supra-phrenic pneumothorax, such as coin percussion note, distant amphoric breath. sounds, musical heart beat, and even a musical fluid tinkle and splash. Two cases of this kind have come under my care. In the ultimate decision as to which side of the diaphragm an air-containing cavity is situated, it must be borne in mind that a subphrenic lesion may raise the arch of the diaphragm as high as the second intercostal space. It may also be bilateral. A case of this kind, which turned out to be a suppurating hydatid of the liver, was under Dr. Habershon's care in Friedenheim. The cavity occupied practically the whole of the front of the thorax below the second right and third left costal cartilages, and its lower margin crossed the epigastrium at the level of the 7th rib. Some information may be obtained as to the situation of a gas-containing cavity under cover of the bony thorax—when effusion has taken place into the pleural cavity—if it is found that the percussion dulness due to the fluid is situated higher up in the chest than the resonance, which is due to the presence of the cavity. The relative positions at which these physical signs are elicited are, as far as they go, evidence that the air-containing cavity is below the diaphragm, and the effusion above it. In practice this is generally the case, but a pneumothorax may be separated off from the rest of the thoracic cavity by adhesions, which prevent the gravitation of fluid to the most dependent part of the pleura. One such case has been under my care, and in this instance, in certain positions, it was easy to obtain the physical signs of an effusion higher up on the chest-wall than the air-containing cavity below. When there is doubt as to whether the lesion is above or below the diaphragm it has been suggested that valuable information can be obtained by puncture with a hollow needle (32); and it has been pointed out that if the needle has been plunged through the diaphragm, its outer end should be raised during inspiration and depressed during expiration (Fürbringer's sign), while if the needle has not passed through the diaphragm, and there is pus in the pleural cavity, it will not move up and down. Again, it has been pointed out that if a collection of matter is struck by the needle above the diaphragm, the flow should be increased during expiration and diminished during inspiration, whilst if the collection is below the diaphragm the reverse should be observed (Pfuhls' sign) (12). Quite apart from the dangers of indiscriminate puncture, the amount of information which is likely to be gained by these signs is small, since the diaphragm is not only liable to be fixed as a direct result of the subphrenic lesion, but it may even move in the inverse direction, as was seen in a case recorded by Devic and Chalier, in which a subphrenic abscess was diagnosed by the *x*-ray screen. The segment of the

diaphragm on the affected side was seen to ascend during inspiration and to descend during expiration, the muscle substance having been infected and softened by the inflammatory processes. The method of attempting to obtain data for a correct diagnosis by puncture with the aspirating needle, as is so often counselled (32), cannot be too strongly deprecated. In the case of a doubtful subphrenic abscess, whether the collection of pus is above or below the diaphragm, there is grave risk of infecting a hitherto unaffected serous cavity, by the passage of the needle either during insertion or withdrawal. The risk of a deliberate exploratory operation conducted so as not to open the pleural cavity before the peritoneal cavity has been closed, or vice versa, is in my experience far less risky than the incautious use of an exploring syringe.

In weighing the value of individual physical signs it must not be forgotten that the diaphragm may be raised as high as the second interspace, either by fluid or by gas; and that the general tendency of encysted collections of fluid below the diaphragm is to raise the heart, inclining the apex-beat, upwards and slightly outwards, rather than to displace that organ sideways away from the lesion, as is the case in large effusions into the pleural cavities. It is sometimes found that in chronic inflammatory lesions in the abdomen, a thickened roll of omentum can be felt through the abdominal wall, stretching across between the umbilicus and the costal arch, as an indurated mass. If this be present in association with other physical signs, which are consistent with the existence of a subphrenic abscess, it may be of value in helping to form a correct diagnosis. The importance of the sign is, however, lessened by the fact that it may be present in malignant disease, and in other forms of chronic peritonitis, apart from any localised collection of pus. It is mainly of importance when associated with a history and with symptoms which suggest that the lesion may be inflammatory, and independent of a previous operation or trauma (cf. Epiploitis, p. 1018).

Leucocytosis, when present, is a valuable indication of suppuration, but it is not always found even when there is a considerable abscess; so that absence of any great increase in leucocytes may be disregarded in summing up the evidence for or against the presence of pus below the diaphragm. A leucocytosis of from 16,000 to 35,000 was present in all the cases at the Boston City Hospital (9), in which subphrenic abscess was a complication of appendicitis.

No useful purpose would be gained by any detailed consideration of the general and constitutional disturbances which may be associated with the lesions under discussion, since they are common to all pus infections. The main points of importance are that the onset may be, on the one hand, that of an acute perforative peritonitis, with high temperature, rapid thready pulse, agonising pain on the lightest pressure over the affected part with possibly filling of the intercostal spaces, and oedema of the chest wall; or on the other that of an insidious malady with little pain, no pyrexia, and indefinite physical signs. As regards

the local conditions, each of the various forms of the disease will give rise to physical signs which are determined by the situation and nature of the initial lesion, the virulence of the poison, the power of resistance of the individual, and the effectiveness of the adhesions which limit the spread of the suppuration.

Prognosis.—Many factors have to be taken into consideration in attempting to form a prognosis. The most important are, the cause of the initial lesion, the method of onset, the extent of the toxaemia, the situation of the abscess—whether intra- or extra-peritoneal, and the effectiveness with which it is isolated by adhesions, the invasion or non-invasion of the thoracic cavity, and lastly, the possibility of operating and the result of the operation. The effect of operation, taking a large number of cases together, is to reduce the mortality by more than half. Thus, of 60 cases recorded by Grüneisen, 40 survived operation, and only 20 died; whilst of 173 cases collected from various sources by Lang (8), of 81 in which no operation was performed only 10 survived; of 14 treated by aspiration, 2 only survived; whilst of 40 operated on, 21 recovered. Grüneisen's (32) cases are as follows:—

Seat of Origin.	Cases.	Cured.	Died.
Appendix	27	18	9
Stomach	9	5	4
Duodenum	1	...	1
Bile-ducts and Gall-bladder . .	2	2	...
Hydatid	3	3	...
Spleen	5	3	2
Pancreas	1	1	...
Perinephric abscess (?) . .	4	2	2
Rib (Caries of) . . .	2	2	...
Pleura (Empyema) . .	4	3	1
Doubtful	2	1	1
	60	40	20

The figures given by Elsberg shew even more favourable results from operation; since of 73 cases, 51 were operated on, and no less than 40 recovered; whilst of the remaining 22 who were not operated on, 18 died. Something may be learnt as to the probable issue of a case from the species of the micro-organisms present in the peritoneal exudate. In Mr. Dudgeon and Mr. Sargent's cases, those in which the *Streptococcus pyogenes* were present were almost invariably fatal, whilst they found the *Bacillus coli* to be less virulent than the *Bacillus pyocyaneus* (cf. *loc. cit.* p. 113).

Treatment.—The treatment of subphrenic abscess is primarily surgical. Unless promptly located and efficiently dealt with by operation it is a very fatal disease, and unless the operation is undertaken before there has been a general infection of the peritoneal cavity, and

before the intrathoracic serous membranes have been invaded, the outlook is wellnigh hopeless. The early recognition of the condition, and the deliberate exploration of the affected part, give the patient the best chance of recovery. As soon as there is reasonable ground for suspecting an abscess below the diaphragm, the seat at which the maximum physical signs are present should be explored. This is not the place to discuss the procedure of the surgical measures to be adopted, but it may perhaps be permissible to say that the results of the operations which I have witnessed, have impressed upon me the importance of shutting off the cavity of the pleura, if it is found to be unaffected, before the parts below the diaphragm are explored. This can be done without difficulty, if the patient's condition is not critical, and if it is possible to allow an interval of two or three days to elapse between the first and second exploratory operations.

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EFFUSIONS INTO THE LESSER SAC OF THE PERITONEUM.—The lesser sac of the peritoneum may be infected from lesions of the stomach, duodenum, pancreas, liver, left kidney and suprarenal, transverse colon, and possibly of the spleen (4), but in inflammatory affections the cavity of the sac is often obliterated before actual perforation into it has occurred. According to Mr. Jordan Lloyd, "pathological distension of the lesser peritoneum gives rise to a tumour in the left hypochondriac, epigastric, and umbilical regions, of a somewhat characteristic shape. It appears to vary from time to time in form and size according to the condition of the overlying stomach, for when the viscus is full of liquid contents, it increases the area of the tumour's dulness, whilst it makes its outline less definite by palpation, and if the stomach be distended with gas the dull area becomes resonant, and apparently the tumour may disappear altogether. The colon always lies below the tumour, and never in front of or above it, as is the case in enlargements of the kidney." When the sac is distended by fluid an area of dulness may be found posteriorly on the left side, owing to the pushing up of the diaphragm (4, 59). Mr. Jordan Lloyd gives three diagrams of cases in which the effusion into the lesser peritoneal cavity was either serum, or serum mixed with blood, but none of them was purulent. In these cases the tumour projected from below the margin of the left costal arch, and extended as an irregularly rounded swelling downwards towards the umbilicus, and to a much less degree into the epigastric and left lumbar regions. After suppuration has occurred not only are the anatomical landmarks liable to be so much altered by adhesions as to make the location of the abscess a matter of great difficulty, but the symptoms are confused by those of the exciting cause, and by the evidences of the acute peritonitis which it sets up.

Suspicion as to the nature of an inflammatory tumour having the characters and situation mentioned above, should be excited when it is found to be resonant on superficial percussion, on account of the presence of the stomach in front of it, and appears to vary in size with the amount of distension of that organ. Otherwise the symptoms in acute cases will be merely those of a suppurative peritonitis, with no special characteristics to distinguish it from other forms of retro-peritoneal abscess.

Whether operated on or not the disease is very fatal. Out of 44 cases alluded to by Michel and Gross, 37 died, and only 7 recovered after operation.

The true nature of the affection seems to have been recognised clinically in two only of the 44 cases quoted by the authors above alluded to; in one, under the care of Spillmann, the situation and nature of the tumour were diagnosed before the operation was undertaken. In the remaining cases all kinds of surmises were made as to the nature of the lesion. They were regarded as cancer of the stomach, gastric ulcer and peritonitis, intestinal obstruction, "epigastric tumour."

The *symptoms* are those of a suppurative peritonitis—and if there are any distinctive physical signs they are those of a retroperitoneal tumour containing fluid.

The *treatment* is purely surgical.

65. Lloyd, Jordan. "Injury to the Pancreas a Cause of Effusions into the Lesser Peritoneal Cavity," *Brit. Med. Journ.*, 1892, ii. 1051.—66. Michel, G., and Gross, G. "Contribution à l'étude des collections purulents de l'arrière cavité des epiploons," *Rev. de Gyn. et de Chir.*, 1904, viii. 45.

Pericolitis and Pericolic Abscess.—Inflammation not infrequently occurs in the connective tissue round the colon, in a manner which is analogous to that which is so common in the right iliac fossa as a result of appendicitis.

Etiology.—Pericolic inflammation may be excited by external violence, such as blows on the abdomen (74A), or by any lesions, mechanical or pathological, in the lumen of the colon, capable of setting up ulceration of the mucous membrane or perforation of the bowel wall. Amongst these may be mentioned: chronic constipation and stercoral ulcers (Power, and Bland-Sutton), ulceration in diverticula from the bowel (Georgi, Beer), and ulcers due to foreign bodies, such as pieces of straw (Cuff), fish-bones, pins, pieces of bone; in fact, any jagged or pointed body which is insoluble, or escapes the action of the digestive fluids, and becomes impacted in the mucous membrane. A tuberculous form of the disease, due to subserous infiltration, has been described by Mr. F. S. Kidd under the name of hyperplastic tuberculous pericolitis, and possibly infection of the subperitoneal tissue may be caused by pyogenetic organisms without any gross breach of the intestinal wall (74, 74A). Two of Mr. Power's cases were on the right side, and he exonerates the appendix of the responsibility of having set up the condition, because it appeared healthy. Similar cases have been recorded by Windscheid. Dr. Hawkins' observations would seem to shew that no appendix can rightly be considered healthy or incapable of causing peritoneal infection, unless it has been examined microscopically, since the micro-organisms which infest it are able to penetrate its walls and infect the peritoneum without any visible breach of surface; also, as has been pointed out, appendix abscess is not infrequently followed by local suppuration at a distance, although no track is left between the two points of suppuration.

In 1893 Mayor gave an account of certain inflammations which he

recognised as occurring in the sigmoid flexure, and called attention to the similarities which they bore to "typhlitis" and "perityphlitis." He describes inflammatory conditions affecting the whole of the thickness of the wall of the bowel in the left iliac fossa, extending either by direct continuity, or through the lymphatics, on the one hand to the peritoneum, and on the other to the cellular tissue. He recognised that they were capable of setting up local inflammation, either within, immediately under, or outside the peritoneum. He also calls special attention to the frequent association of constipation with this condition. These cases are of interest and importance, because there is often no clue at the bedside to the cause of the lesion; and if the inflammation is low down in the colon, there may be symptoms of obstruction, simulating those which result from new growth (72A, 74A). If an abscess form and discharge into the bowel, the tumour may, as Mr. Bland-Sutton observes, spontaneously disappear, after having been declared to be an irremovable cancer; and in spite of the gloomiest prognostications the patient may rapidly recover and live to mock at his advisers (69, 74A).

Pericolitis is most likely to occur in those situations in which the mechanical efficiency of the bowel is least, namely, at the hepatic, splenic, or sigmoid flexures. If the lesion is limited to the ascending colon, the appendix is obviously liable to get the discredit; if it is in the sigmoid flexure it may be impossible to differentiate it clinically from malignant disease; in any part of the colon it may be mistaken for neoplasm, tuberculosis, or actinomycosis. Besides the authors already mentioned, Drs. Rolleston (*pericolitis sinistra*), Newton, Tavel, and others have recorded cases, and I am indebted to the writings of the two former observers for some of the references given below. Pericolitis, whether it originates in the mucous membrane, submucous or subperitoneal tissue, or in the peritoneum itself, may end in resolution, fibrosis, or suppuration. If the inflammation become chronic, it tends to the formation of adhesions between neighbouring organs. These adhesions contract, increase in thickness, and form bands which may give rise to kinking, narrowing, and fixation of the bowel. If suppuration occurs, an intra- or extra-peritoneal abscess may result, which may become encysted, or, if not relieved by operation, may rupture into the bowel, the peritoneal cavity, or even externally.

The *symptoms* vary considerably according to the seat of origin and nature of the inflammation. There may be constant or intermittent fever; local tenderness, especially on pressure, radiating to the back; disturbance in the functions of the bowel—such as constipation or diarrhoea, with or without mucus and blood in the motions. The latter symptoms vary greatly and depend primarily on the extent to which the mucous membrane has been involved, and whether or no there has been ulceration.

The *physical signs* need not be described in detail, as they are those which are common to many localised abdominal tumours accompanied by inflammation. Their variations depend mainly on the extent of

the inflammation, the amount of pus which has been formed, and the interference with the functions of the bowel and of neighbouring viscera which results from the presence of adhesions.

If suppuration has occurred the *treatment* is surgical. Evacuation of the abscess is the only safe method of giving relief. If the symptoms are indefinite, the tumour ill defined, and without signs of suppuration, palliative measures — laxatives, internal lubricants, such as oil, or emulsions of one of the vaseline preparations, combined with enemas containing oil—may be used with advantage. If there is evidence of ulceration of the lower bowel, washing out with copious warm enemas of normal saline solution or gruel will give much temporary relief.

Abscesses in Appendices Epiploicae.—Mr. Bland-Sutton has called attention to a variant on the above class of cases, which he has noticed and described, namely, pericolic abscesses limited to an epiploic appendage. "It is," he says, "clear from a consideration of the normal anatomy of the colon that there are portions of its circumference where a sharp foreign body could penetrate and escape into the general peritoneal cavity, and the thinner or more emaciated the person the greater is the arc of the gut which would allow this form of penetration. In a fat person, on the other hand, although there is a short arc where it could be easily perforated into the general peritoneal cavity, there is a far greater extent of its circumference protected by fat, into which the escaping body would be more probable to find its way, and in a certain proportion of cases the penetrating body would lodge in an adjacent epiploic appendix. The fatter the patient the less likely is the foreign body to penetrate into the general peritoneal cavity, and the greater is its chance of entering an epiploic appendix."

From the foregoing account of abscess in an appendix epiploica, it is hardly to be expected that the precise nature of the affection will often be correctly diagnosed before operation or autopsy. Where so many causes may give rise to symptoms closely resembling one another, the diagnosis must in most instances rest rather on surmise and a recognition of all the possibilities, than on logical deduction. Such cases shew the necessity, when estimating the gravity of a tumour attached to the bowel, of taking into account the possibility of its being inflammatory, and the recognition of such conditions as those which have been described may help to indicate a rational line of treatment, which gives hope of ultimate success. It may also serve to throw light on the spontaneous disappearance of tumours which have been, on clinical evidence alone, regarded as malignant. If suppuration has occurred, the treatment is purely surgical, but in less pronounced cases palliative measures, such as have been suggested in pericolitis, may be adopted.

67. Beer, E. "Some Pathological and Clinical Aspects of Acquired (false) Diverticula of the Intestine," *Amer. Journ. Med. Sci.*, 1904, cxxviii. 135, *q.v.* for references. — 67A. Beuttner, O. "Un Cas de péricolite (post-appendiculaire), Laparotomie, Guérison," *Rev. méd. de la Suisse Rom.*, Genève, 1905, xxv. 132.—

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Med., 1882, xxx. 265.

Epiploitis, or inflammation of the great omentum, is a localised form of peritonitis, which is of special importance because it has so frequently been found to follow appendicitis and operation for hernia, and, if the cause is not recognised, may easily be mistaken for carcinoma or tuberculosis of the peritoneum. Schnitzler collected 24 cases previous to 1900, and himself gives four others, every one of which was the sequel of an operation for hernia. Most of them followed the use of silk ligatures applied to the omentum. Attention seems to have been most specially called to the subject by Championnière in [illegible].

The pathological anatomy of epiploitis is that of an infective peritonitis, and need not be given in detail.

The *symptoms* are those of a more or less acute peritonitis limited to the great omentum. Great variations are found in the symptoms, physical signs, and constitutional disturbances, according as the condition is acute or chronic, localised or diffuse, adhesive or suppurative. If the onset is acute or subacute, there may be pyrexia, vomiting, abdominal pain, greatly increased by pressure, with all the other symptoms of peritonitis. If the onset is insidious, the symptoms more closely resemble those which are found in malignant and tuberculous diseases of the peritoneum, viz. increasing difficulty with the bowels, moderate pain, and distension of the abdomen.

On physical examination, if the muscular resistance permit it, a more or less nodular irregular swelling may be made out, and if suppuration has already taken place areas of fluctuation may be detected. The course of the disease may be acute or chronic, lasting even possibly for years, and it may end in resolution, if there has been no formation of pus. If suppuration has occurred the abscess may rupture into one of the abdominal viscera, or into the general cavity of the peritoneum. Even if pus does not form, grave consequences may result from adhe-

sions with the neighbouring coils of bowel, and consequent intestinal obstruction.

The *treatment*, if the symptoms are not urgent, should be symptomatic. Previous to suppuration the inflammation may subside. If abscesses form they must be treated surgically; and if possible the cause of irritation must be removed.

T. D. Acland.

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